V. 13.

Library

of the

Academy of Medicine

Toronto.

17386

Presented by

American Otological Society.

1926

TRANSACTIONS

OF THE

AMERICAN OTOLOGICAL SOCIETY,

FORTY-SIXTH ANNUAL MEETING.

HOTEL RALEIGH,

WASHINGTON, D. C.

MAY 6 and 7, 1913.

VOLUME XIII. PART I.

LIBRARY
JUL 1 8 2002
UNIVERSITY OF TORONTO

PUBLISHED BY THE SOCIETY.
MERCURY PUBLISHING COMPANY, PRINTERS.
NEW BEDFORD, MASS.
1913.

Extra copies may be had by addressing the secretary, Dr. John B. Rae, 247 West 70th Street, New York, N. Y., or Mercury Publishing Company, 112 Union Street, New Bedford, Mass.

CONTENTS.

See also p. 255, 413

	PAGE
Members of the American Otological Society	5
Minutes of the Forty-Sixth Annual Meeting	10
Presentation of Instruments. A Forceps with Long Handles. By Emil Amberg, Detroit, Mich	17
The Prevention of Deafness. By G. Hudson-Makuen, Philadelphia, Pa	18
Discussion	26
Report of a Case of Tempero-Sphenoidal Abscess of Otitic Origin. Operation. Recovery. By James F. McCaw, M. D., Watertown, N. Y	29
Discussion	32
A Skull Trephined for Mastoid Caries and Lateral Sinus Thrombosis by the Late Dr. Samuel Ashhurst of Philadelphia Some Forty Years Ago. By B. Alexander Randall, M. D., Philadelphia, Pa	37
The Pathology of the Labyrinth as Shown in Microscopic Sections (Stereopticon Demonstration). By Max A. Goldstein, M. D., St. Louis, Mo	40
Report of Case of Suppurative Labyrinthitis. Recovery without Operation. Comments on Diagnosis and Indications for Treatment. By O. A. M. McKimmie, M. D., Washington, D. C	52
Discussion	56
Report of Two Cases of Cerebrospinal Meningitis Presenting no Central Nervous Phenomena Until Shortly Before Death. By T. Passmore Berens, M. D., New York, N. Y.	59
Abscess of Frontal Lobe of the Brain of Otitic Origin, with Exhibition of Specimens. By T. Passmore Berens, M. D., New York, N. Y	66
Discussion	87
The Protective Mastoid Operation; An Operation of Election. By W. Sohier Bryant, A. M., M. D., New York, N. Y.	89
Preservation of the Antral Capsule in Operations for Uncomplicated Acute Suppurative Processes of the Mastoid. By Norval H. Pierce, M. D., Chicago, Ill	99
Discussion	103

Further Observations on the Technique in the After-Care of the Radical Mastoid Operation. By Philip Hammond, M. D., Boston, Mass. 107
Discussion 114
Influenzal Otitis. By S. MacCuen Smith, M. D., Philadelphia, Pa. 119
Discussion 125
A Brief Consideration of Certain Recent Views Regarding Otosclerosis. By Thomas J. Harris, New York, N. Y. . 127
Ocular Manifestations in Nasal and Aural Diseases which Probably Indicate Involvement of the Sympathetic Nervous System. By William H. Haskin, M. D., New York, N. Y. 144
Discussion 171
When to Operate on the Labyrinth in Labyrinth Infection Secondary to Purulent Otitis Media. By G. E. Shambaugh, Chicago, Ill. 173
Discussion 177
A Report of Three Cases of Otitic Meningitis, Treated by Drainage of the Cisterna Magna. By Edward Bradford Dench, M. D., New York, N. Y. 180
Discussion 184
Exposure and Curettement of the Attic Combined with a Modified Blood Clot as Factors in Promoting Rapid Mastoid Healing. By Hugh B. Blackwell, M. D., New York, N. Y. 190
Discussion 194
A Report of the Results of Seven Cases Operated Upon by Mr. Heath. By Edward M. Plummer, M. D., and Harris P. Mosher, M. D., Boston, Mass. 196
Discussion 232
The Use of Vaccines After the Mastoid Operation. By William C. Braislin, M. D., Brooklyn, N. Y. 234
Discussion 239
The Implantation of Cartilage for the Prevention and Correction of Deformity of the Auricle. By Harris Peyton Mosher, M. D., Boston, Mass. 240
The Value of Naso-Pharyngeal Surgery in the Treatment of Chronic Exudative Otitis Media. By H. O. Reik, M. D., Baltimore, Md. 245

MEMBERS

OF THE

AMERICAN OTOLOGICAL SOCIETY.

Names of deceased members while in membership in full face.

HONORARY MEMBERS.

Prof. A. Graham Bell	1336 19th St	Washington, D. C.
Dr. Albert H. Buck	Hewetts	Long Island, N. Y.
Dr. E. H. Clarke.		
Dr. John Green	2670 Washington Ave	St. Louis, Mo
Dr. J. Orne Green	267 Newbury St	Boston, Mass
Dr. Edw. L. Holmes.		
Dr. Hermann Knapp.		
Sir William Macewen	3 Woodside Crescent	Glasgow, Scotland
Dr. Arthur Mathewson		Washington, D. C
Prof. A. F. Mayer.		
Prof. Adam Politzer	1 Gongazagesse Sq	Vienna, Austria
Dr. Thomas R. Pooley	40 East 41st St	New York N. Y.
Dr. Jonathan S. Prout		Garden City, L. I.
Dr. H. Luc	54 Rue de Varenne	Paris, France
Dr. E. Williams.		

MEMBERS.

Dr. Frank W. Abbott.		
Dr. Alpheus E. Adams	44 Grand St	Newburg, N. Y.
Dr. John L. Adams	38 East 51st St	New York, N. Y.
Dr. C. R. Agnew.		
Dr. W. F. Akin.		
Dr. Henry A. Alderton	142 Clinton St	Brooklyn, N. Y.
Dr. H. P. Allen.		
Dr. Adolph Alt	316 Metropolitan Bldg	St. Louis, Mo
Dr. Alfred M. Amadon*	543 Boylston St	Boston, Mass
Dr. Emil Amberg*	270 Woodward Ave	Detroit, Mich
Dr. Joseph A. Andrews		Santa Barbara, Cal
Dr. Stephen C. Ayres	4 West 7th St	Cincinnati, Ohio
Dr. Gorham Bacon	47 West 54th St	New York, N. Y.
Dr. Robert Barclay	3894 Washington Boulevard	St. Louis, Mo
Dr. John F. Barnhill	Rms. 404-411 Pennway Bldg.	Indianapolis, Ind

Dr. Edwin W. Bartlett.....422 Jefferson St.....Milwaukee, Wis.....
Dr. T. Passmore Berens....35 Park Ave.....New York, N. Y.....
Dr. Herbert S. Birkett.....252 Mountain St.....Montreal, Canada.....
Dr. Hugh B. Blackwell*...148 West 58th St.....New York City.....
Dr. Clarence J. Blake.....226 Marlborough St.....Boston, Mass.....
Dr. Frank J. Blodgett.....140 West 72nd St.....New York, N. Y.....
Dr. Charles R. C. Borden*..484 Beacon St.....Boston, Mass.....
Dr. William C. Braislin....556 Washington Ave.....Brooklyn, N. Y.....
Dr. Joseph H. Bryan.....818 17th St. N. W.....Washington, D. C.....
Dr. W. Sohier Bryant.....19 West 54th St.....New York, N. Y.....
Dr. Ernest U. Buckman....99 South Franklin St.....Wilkes Barre, Pa.....
Dr. Charles H. Burnett.
Dr. Ralph Butler.....1824 Chestnut St.....Philadelphia, Pa.....
D. R. Bishop Canfield.....Ann Arbor, Mich.....
Dr. Franklin P. Capron.....118 Angell St.....Providence, R. I.....
Dr. James B. Clemens.....603 5th Ave.....New York, N. Y.....
Dr. Cornelius G. Coakley...53 West 56th St.....New York, N. Y.....
Dr. William Cowen.....35 East 60th St.....New York, N. Y.....
Dr. Charles N. Cox.....257 Jefferson Ave.....Brooklyn, N. Y.....
Dr. Eugene A. Crockett.....298 Marlborough St.....Boston, Mass.....
Dr. J. Frank Crouch.....513 North Charles St.....Baltimore, Md.....
Dr. John P. Davidson.
Dr. Ewing W. Day.....1005 Westinghouse Bldg....Pittsburg, Pa.....
Dr. Edward B. Dench.....15 East 53rd St.....New York, N. Y.....
Dr. J. H. Dix.
Dr. Ernest E. Doble.....52 Cross St.....Quincy, Mass.....
Dr. Arthur B. Duel.....27 East 57th St.....New York, N. Y.....
Dr. John Dunn*.....314 East Franklin St.....Richmond, Va.....
Dr. E. Dwyer.
Dr. Wells P. Eagleton.....15 Lombardy St.....Newark, N. J.....
Dr. E. P. Ely.
Dr. Joseph B. Emerson.....40 East 41st St.....New York, N. Y.....
Dr. George F. Fiske.....100 State St.....Chicago, Ill.....
Dr. Edw. Fridenberg.
Dr. Percy Fridenberg.....60 East 58th St.....New York, N. Y.....
Dr. Harry Friedenwald*....1029 Madison Ave.....Baltimore, Md.....
Dr. Blancome E. Fryer.
Dr. Max A. Goldstein*.....3858 Westminster Place...St. Louis, Mo.....
Dr. Emil Gruening.....36 East 57th St.....New York, N. Y.....
Dr. George W. Hale.....Wilcox Building.....Nashville, Tenn.....
Dr. Thomas H. Halsted.....831 University Block.....Syracuse, N. Y.....
Dr. Philip Hammond.....483 Beacon St.....Boston, Mass.....
Dr. Herbert Harlan.....516 Cathedral St.....Baltimore, Md.....
Dr. Thomas J. Harris.....104 East 40th St.....New York, N. Y.....
Dr. David Harrower.....9 Elm St.....Worcester, Mass.....

Dr. N. Darrell Harvey.....114 Watermann St.....Providence, R. I.....
Dr. William H. Haskin.....40 East 41st St.....New York, N. Y.....
Dr. Neil J. Hepburn.....317 West 23rd St.....New York, N. Y.....
Dr. James Hewitt.
Dr. Christian R. Holmes....8 East 8th St.....Cincinnati, Ohio.....
Dr. Edgar M. Holmes*.....531 Beacon St.....Boston, Mass.....
Dr. Erastus E. Holt.....723 Congress St.....Portland, Me.....
Dr. Frank T. Hopkins*....182 West 58th St.....New York City.....
Dr. Thomas Hubbard*.....515 Nicholas Bldg.....Toledo, O.....
Dr. Frederick L. Jack.....215 Beacon St.....Boston, Mass.....
Dr. Walter B. Johnson.....170 Broadway.....Paterson, N. J.....
Dr. Richard Jordan.....165 West 58th St.....New York, N. Y.....
Dr. Joseph A. Kenefick.....78 East 54th St.....New York, N. Y.....
Dr. B. R. Kennon.....64 Granby St.....Norfolk, Va.....
Dr. Philip D. Kerrison.....58 West 56th St.....New York, N. Y.....
Dr. Gordon King.
Dr. Charles J. Kipp.
Dr. D. Braden Kyle.....1517 Walnut St.....Philadelphia, Pa.....
Dr. J. W. Lawton.
Dr. George A. Leland.....354 Commonwealth Ave....Boston, Mass.....
Dr. John C. Lester.....432 Madison Ave.....New York, N. Y.....
Dr. Robert Lewis .....48 West 40th St.....New York, N. Y.....
Dr. W. S. Little.
Dr. Hanau W. Loeb*.....537 N. Grand Ave.....St. Louis, Mo.....
Dr. James E. Logan.....1208 Wyandotte St.....Kansas City, Mo.....
Dr. Francis B. Loring.....1420 K St., N. W.....Washington, D. C.....
Dr. David B. Lovell.....32 Pearl St.....Worcester, Mass.....
Dr. C. J. Lundy.
Dr. Stephen H. Lutz*.....284 Hancock St.....Brooklyn, N. Y.....
Dr. G. Hudson-Makuen*....1627 Walnut St.....Philadelphia, Pa.....
Dr. George B. McAuliffe....616 Madison Ave.....New York, N. Y.....
Dr. James F. McCaw.....Bank & Loan Bldg.....Watertown, N. Y.....
Dr. Read J. McKay.....9th & Market Sts.....Wilmington, Del.....
Dr. James F. McKernon....62 West 52nd St.....New York, N. Y.....
Dr. O. A. M. McKimmie*..1330 Mass. Ave. N. W.....Washington, D. C....
Dr. Cyrus S. Merrill.....27 South Hawk St.....Albany, N. Y.....
Dr. Horace J. Miller.
Dr. W. W. Moreland.
Dr. Henry L. Morse.....112 Marlborough St.....Boston, Mass.....
Dr. Harris P. Mosher*.....828 Beacon St.....Boston, Mass.....
Dr. Willis B. Moulton*.....180 State St.....Portland, Me.....
Dr. Robert C. Myles.....46 West 38th St.....New York, N. Y.....
Dr. James E. H. Nichols.
Dr. Henry D. Noyes.
Dr. J. F. Noyes.

Dr. Francis R. Packard....304 South 19th St.....Philadelphia, Pa.....
Dr. John R. Page*.....127 East 62d St.....New York, N. Y.....
Dr. Charles E. Perkins*....105 West 77th St.....New York, N. Y.....
Dr. Wendell C. Phillips.....40 West 47th St.....New York, N. Y.....
Dr. Norval H. Pierce.....22 E. Washington St.....Chicago, Ill.....
Dr. Edward M. Plummer....5 Adams St.....Charlestown, Mass....
Dr. Oren D. Pomeroy.
Dr. Barton H. Potts.
Dr. John B. Rae.....247 West 70th St.....New York, N. Y.....
Dr. B. Alexander Randall...1717 Locust St.....Philadelphia, Pa.....
Dr. Wm. Rankin, Jr.
Dr. J. M. Ray.....Atherton Building.....Louisville, Ky.....
Dr. Richard A. Reeve.....48 East Bloor St.....Toronto, Canada.....
Dr. Henry O. Reik.....506 Cathedral St.....Baltimore, Md.....
Dr. W. Scott Renner.....341 Linwood Ave.....Buffalo, N. Y.....
Dr. George L. Richards.....124 Franklin St.....Fall River Mass.....
Dr. Huntington Richards.. St. Paul's School.....Concord, N. H.....
Dr. John D. Richards*.....44 West 49th St.....New York, N. Y.....
Dr. Charles W. Richardson..1317 Connecticut Ave.....Washington, D. C.....
Dr. Stephen O. Richey.....732 17th St.....Washington, D. C.....
Dr. Charles E. Rider.
Dr. F. W. Ring.
Dr. Henry W. Ring.....187 Church St.....New Haven, Conn....
Dr. Edmund C. Rivers.....1632 Welton St.....Denver, Col.....
Dr. Arthur W. de Roaldes...624 Gravier St.....New Orleans, La.....
Dr. Walter Roberts.....1732 Spruce St.....Philadelphia, Pa.....
Dr. William K. Rogers....88 East State St.....Columbus, Ohio.....
Dr. D. B. St. John Roosa.
Dr. John D. Rushmore.....129 Montague St.....Brooklyn, N. Y.....
Dr. T. Laurance Saunders*.43 West 50th St.....New York City.....
Dr. Wm. W. Seely.
Dr. Ralph W. Seiss.....255 South 17th St.....Philadelphia, Pa.....
Dr. S. Sexton.
Dr. George E. Shambaugh..People's Gas Bldg.....Chicago, Ill.....
Dr. John B. Shapleigh.....Grand & Washington Aves..St. Louis, Mo.....
Dr. Warren S. Shattuck, Jr.160 Clinton St.....Brooklyn, N. Y.....
Dr. Henry L. Shaw.
Dr. John E. Sheppard.....130 Montague St.....Brooklyn, N. Y.....
Dr. Burt R. Shurly*.....32 Adams Ave. W.....Detroit, Mich.....
Dr. E. Terry Smith.....70 Cove St.....Hartford, Conn.....
Dr. S. MacCuen Smith.....1429 Spruce St.....Philadelphia, Pa.....
Dr. Sargent F. Snow.....707 University Block.....Syracuse, N. Y.....
Dr. Edmund D. Spear.....100 Boylston St.....Boston, Mass.....
Dr. Horatio N. Spencer.....2725 Washington Ave.....St. Louis, Mo.....
Dr. Selden Spencer*.....2725 Washington Ave.....St. Louis, Mo.....

Dr. Frank Sprague.
Dr. Franklin M. Stephens...71 West 49th St.....New York, N. Y.....
Dr. George T. Stevens.....40 East 41st St.....New York, N. Y.....
Dr. Edward Stieren.....Westinghouse Building....Pittsburg, Pa.....
Dr. Samuel B. St. John.
Dr. George Strawbridge....202 South 15th St.....Philadelphia, Pa.....
Dr. Joseph A. Stucky*.....Lexington, Ky.....
Dr. Theron Y. Sutphen....999 Broad St.....Newark, N. J.....
Dr. Lewis H. Taylor.....82 South Franklin St.....Wilkes Barre, Pa.....
Dr. Samuel Theobald.....Cathedral & Howard Sts....Baltimore, Md.....
Dr. William Thomson.
Dr. Robert Tilley.
Dr. J. J. Thomson*.....40 West 47th St.....New York City.....
Dr. Andrew Timberman*...112 East Broad St.....Columbus, Ohio.....
Dr. J. J. B. Vermyne.
Dr. Ernest de Wolfe Wales..423 Board of Trade Bldg.. Indianapolis, Ind.....
Dr. D. Harold Walker.....390 Commonwealth Ave....Boston, Mass.....
Dr. David Webster.....308 Madison Ave.....New York, N. Y.....
Dr. Joseph A. White.....200 East Franklin St.....Richmond, Va.....
Dr. Edward Wheelock.....26 Gibbs St.....Rochester, N. Y.....
Dr. Fred Whiting.....19 West 47th St.....New York, N. Y.....
Dr. Frederick M. Wilson...834 Myrtle Ave.....Bridgeport, Conn.....
Dr. John Gordon Wilson*..104 S. Michigan Ave.....Chicago, Ill.....
Dr. Hiram Woods.....842 Park Ave.....Baltimore, Md.....

*Associate.

American Otological Society.

The forty-sixth annual meeting of the American Otological Society was held at the Hotel Raleigh, Washington, D. C., on Tuesday and Wednesday, May 6th and 7th, 1913.

The following registered during the meeting:

Members.

J. L. ADAMS
JOHN F. BARNHILL
T. P. BERENS
HERBERT S. BIRKETT
WM. C. BRAISLIN
JOSEPH H. BRYAN
W. SOHIER BRYANT
RALPH BUTLER
C. G. COAKLEY
E. A. CROCKETT
J. FRANK CROUCH
EWING W. DAY
EDWARD B. DENCH
ARTHUR B. DUEL
GEORGE F. FISKE
PERCY FRIDENBERG
EMIL GRUENING
THOS. H. HALSTED
PHILIP HAMMOND
HERBERT HARLAN
THOS. J. HARRIS
DAVID HARROWER
N. DARRELL HARVEY
WM. H. HASKIN
C. R. HOLMES
ERASTUS E. HOLT
FREDERICK L. JACK
RICHARD JORDAN
JOSEPH A. KENEFICK
B. R. KENNON
PHILIP D. KERRISON
D. BRADEN KYLE
GEORGE A. LELAND
JOHN C. LESTER
JAMES E. LOGAN
FRANCIS B. LORING
DAVID B. LOVELL
GEO. B. McAULIFFE
JAMES F. McCAW
JAMES F. McKERNON
ROBERT C. MYLES
FRANCIS R. PACKARD
WENDELL C. PHILLIPS
NORVAL H. PIERCE
JOHN B. RAE
B. ALEX. RANDALL
J. M. RAY
HENRY O. REIK
W. SCOTT RENNER
GEORGE L. RICHARDS
STEPHEN O. RICHEY
HENRY W. RING

WALTER ROBERTS
GEORGE E. SHAMBAUGH
S. MacCUEN SMITH
SARGENT F. SNOW
E. TERRY SMITH
LEWIS H. TAYLOR
JOSEPH A. WHITE
HIRAM WOODS
THERON Y. SUTPHEN

Associate Members.

EMIL AMBERG
HUGH B. BLACKWELL
R. B. CANFIELD
MAX A. GOLDSTEIN
HANAU W. LOEB
STEPHEN H. LUTZ
HARRIS P. MOSHER
O. A. M. McKIMMIE
JOHN D. RICHARDS
JOSEPH A. STUCKY
J. J. THOMSON
JOHN GORDON WILSON
G. HUDSON-MAKUEN

Guests.

FRANCIS WELCH
F. E. HOPKINS
F. E. KETTRIDGE
ALFRED BRAUN
E. R. LEWIS
HOWARD PYFEE
R. C. LYNCH
W. E. SAUER
JOSEPH BECK
C. COLTER CHARLTON
CHAS. C. AMERMAN
H. M. FISH
HILL HASTINGS
SAMUEL J. KOPETZKY
C. W. WALDRON
JAMES DWYER
GEORGE E. CARROLL
M. M. SAVAGE
E. REID RUSSELL
J. B. GREENE
R. J. TEAGUE
R. E. BRAWLEY
WM. L. BALLINGER
F. E. SONDERN
J. W. MURPHY
F. ALLPORT

Executive Session.

Tuesday Morning, May 6th, 10 A. M.

The meeting was declared open when the President, Dr. James F. McKernon, briefly thanked the members for his election.

The President appointed Dr. Ewing W. Day, Dr. S. MacCuen Smith and Dr. George F. Fiske to act as the Business Committee.

The minutes of the forty-fifth annual meeting were read and approved.

The report of the Committee on Membership was received. On motion the following gentlemen were elected to membership in the Society: Dr. Chas. E. Perkins, New York City; Dr. E. M. Holmes, Boston, Mass.; Dr. C. R. C. Borden, Boston, Mass.; Dr. R. Bishop Canfield, Ann Arbor, Mich.; Dr. John R. Page, New York City.

On motion the following were carried over for one year: Dr. A. P. Voislawsky, New York City; Dr. George H. Powers, Boston, Mass.; Dr. George L. Toby, Boston, Mass.; Dr. R. Johnson Held, New York City; Dr. Edw. Blair Sutphen, Morristown, N. J.

A communication was read by the Secretary with reference to co-operation of the Society in a movement to provide suitable memorials for Lister. On motion Drs. Randall, Dench and Shambaugh were appointed a committee to act on this matter.

A communication on "Internationalism" was read by the Secretary and referred to the same committee.

A communication with reference to holding the 1915 meeting at San Francisco was read and referred to the Business Committee.

A communication was read suggesting the change of date of meeting to the week between Christmas and New Years. This was also referred to the Business Committee.

The report of the Treasurer was read and on motion was approved.

Scientific Session.

Dr. Amberg presented an angled forceps especially designed to assist in mastoid operations and mastoid dressings.

Dr. Canfield presented an instrument for deep suturing in the nose.

Papers were read by the following: Dr. Hudson-Makuen, Dr. McCaw, Dr. Randall, Dr. Goldstein, Dr. McKimmie and Dr. Berens.

Executive Session.

Wednesday, May 7th, 9 A. M.

The President appointed as delegate and alternate respectively, to the International Congress in London, Dr. Edward B. Dench, New York City, and Dr. H. O. Reik, Baltimore, Md.

Excuses for absence were presented by Dr. Joseph A. Andrews, Dr. Arthur W. de Roaldes, Dr. Alfred M. Amadon and Dr. Thomas Hubbard.

On motion the excuses were accepted.

The Society then adjourned to Scientific Session when papers were read by Dr. Berens, Dr. Bryant, Dr. Pierce, Dr. Hammond, Dr. MacCuen Smith, Dr. Harris and Dr. Haskin.

Executive Session.

Wednesday Afternoon, May 7th, 1913.

Report of the Business Committee. The Business Committee presented the following nominations:

President—James F. McKernon, New York City.

Vice-President—Eugene A. Crockett, Boston, Mass.

Secretary and Treasurer—John B. Rae, New York City.

Committee on Membership—Dr. E. B. Dench, New York City, to serve one year; Geo. E. Shambaugh, Chicago, Ill., to serve two years; H. S. Birkett, Montreal, Canada, to serve three years.

Committee on Publication—B. Alexander Randall, Philadelphia, Pa.; Arthur B. Duel, New York City; the Secretary, *ex officio.*

On motion the report was accepted and the above named officers declared elected.

The following candidates were nominated for membership: Dr. Benjamin D. Parish, Philadelphia, Pa.—Proposed by Dr. B. Alexander Randall; seconded by Dr. Ralph Butler. Dr. Clifton Miller, Richmond, Va.—Proposed by Dr. Joseph A. White; seconded by Dr. B. R. Kennon. Dr. Wil-

liam E. Sauer, St. Louis, Mo.—Proposed by Dr. John F. Barnhill; seconded by Dr. B. Alexander Randall. Dr. Walter A. Wells, Washington, D. C.—Proposed by Dr. F. R. Packard; seconded by Dr. Geo. L. Richards. Dr. Eugene R. Lewis, Dubuque, Ia.—Proposed by Dr. Norval H. Pierce; seconded by Dr. Wendell C. Phillips.

The Business Committee made an unfavorable report with reference to 1915 meeting at San Francisco. On motion this was accepted. The Business Committee reported unfavorably with regard to the change of time of meeting. On motion this was indorsed by the Society.

The President appointed Dr. Crockett, Dr. Shambaugh, the President and the Secretary, a committee of time and place for the next meeting. On motion of Dr. Dench it was decided that the meeting be held no later than June 25th.

Dr. Richards reported the death of Mr. Lane, connected with the Mercury Publishing Company, who had for many years taken a special interest in the publication of the transactions of the Society.

Dr. Richards, Dr. Jack and Dr. Plummer were appointed a committee to give appropriate expression to the appreciation and regret of the society.

This committee later presented the following:

APPRECIATION.

The American Otological Society having learned with sorrow of the death of Mr. Willard Lane, of the Mercury Publishing Co., wishes to put on record its appreciation of the labor which Mr. Lane gave to the preparation of its annual volume for so many years. The volume was first published in New Bedford under his supervision in 1879, when the late Dr. Vermyne was secretary of the Society, and from that date to this the volumes bear witness to the keen personal interest which Mr. Lane took in their production, which came to him to be a matter of professional pride rather than of business profit.

G. L. RICHARDS,
F. L. JACK,
C. M. PLUMMER,
Committee.

Dr. Richards proposed that the writers of papers be requested to send with their papers a brief abstract with the view of having these abstracts appear in the general medical journals at appropriate times throughout the year. The society approved of this idea and on motion the matter was referred to the Publication Committee with power.

On motion of Dr. Phillips the Secretary was instructed to insert in the program the time of the different executive sessions to be held during the meeting of the society and the different matters to be discussed as far as possible at these sessions.

On motion of Dr. Shambaugh it was resolved to follow the pre-arranged program as closely as possible.

On motion of Dr. Crockett it was resolved that the indiscriminate granting of operating and other privileges to visiting foreigners was deemed unwise by this society.

A communication was read from the American Gynecological Society requesting the appointment of a committee to work in conjunction with similar committees to bring about the education of the public as to the benefit of the early recognition of cancer.

Dr. Richardson, Dr. Shambaugh and Dr. C. R. Holmes were appointed on this committee.

On motion the thanks of the Society were extended to Dr. Francis B. Loring, of Washington, for the excellent arrangements he had made for this meeting.

On adjournment of the Executive Session, papers were read by the following:

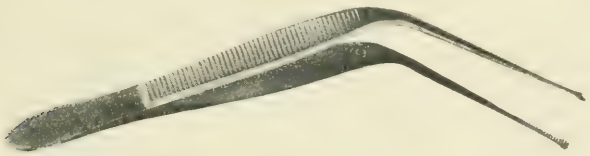
Dr. Shambaugh, Dr. Dench, Dr. Blackwell, Dr. Mosher, Dr. Braislin and Dr. Mosher.

The Society then adjourned.

PRESENTATION OF INSTRUMENTS. A FORCEPS WITH LONG HANDLES.

By EMIL AMBERG, Detroit, Michigan.

In 1899 I published a forceps which differs from an ordinary ear forceps by having long handles. The figure accompanying the publication in 1899 gives an idea of the shape of the forceps, also of the blunt endings. Recently I have had made a slightly stronger forceps along the same lines and have the endings still more rounded out according to the endings of the forceps of Mueller.



In my opinion, this forceps is especially valuable for two reasons: *First,* the long handles make it possible that the hand of the assistant, e. g., in a mastoid operation, does not interfere so much as it would if he uses a forceps with an ordinary handle. *Second,* the whole hand can be utilized in holding the instrument. If desired, the shanks may be made longer or stronger, or both. It may be added that the inner parts of the ends of the shanks are either smooth or grooved. The first may be more useful in doing dressings. The accompanying illustration depicts the instrument in about half its size.

THE PREVENTION OF DEAFNESS.

By G. HUDSON-MAKUEN, Philadelphia, Pa.

Someone has said that the duty of the physician is threefold: (1) to prevent; (2) to cure; and (3) to alleviate. Applying this principle to the subject of deafness, it would seem that we have reversed the order of the three duties, and our efforts have been: (1) to alleviate; (2) to cure; (3) to prevent.

Someone has also well said that the time to cure deafness is before it begins, a seemingly paradoxical statement and one implying that the affection when thoroughly established is incurable. The prevention of deafness, therefore, is a subject toward which the attention of the modern otological world is rapidly turning, and one which the progressive general practitioner of medicine can no longer ignore.

One of the difficulties arising in any discussion of this subject is to make a satisfactory etiological classification of it. The assigned causes for deafness are so numerous and varied that it is difficult even to group them. In a recent report issued by the United States Bureau of Census in which 89,287 cases of so-called deaf mutism were returned, over 500 different causes were assigned for the condition.

The classification usually made in dealing with the subject, namely congenital and acquired deafness, is not altogether satisfactory, because it is difficult to differentiate between the two conditions and many of the recognized causal factors are common to both congenital and acquired deafness. Similar objections arise, however, in any classification of the subject, but the one that appears to be the most satisfactory for my purpose is the following:

CLASSIFICATION OF DEAFNESS.

- Deafness
  - Inherited
    - Gross Physical Anomolies, Congenital—Total deafness, deaf mutism.
    - Minor Physical Anomolies, Congenital
      - Partial Deafness of Infancy.
      - Tendency to become deaf in childhood and adult life.
  - Acquired
    - Diseases of the Ear
      - Primary, originating in the ear.
      - Secondary, resulting from systemic diseases.
    - Injuries to the Ear
      - Falls, Blows, Explosions, Burns, Scalds.

The great objection to this classification is the fact that it is extremely difficult to entirely eliminate heredity as a causal factor even in many of the so-called acquired cases.

Deafness, let it be remembered, is not a disease, but it is the result of certain physical conditions which are either inherited or acquired.

The inherited conditions which result in deafness are for the most part congenital, and they appear either as gross physical anomalies of the organs of hearing such as are productive of the profound deafness of deaf mutism, or as minor physical anomalies not necessarily confined to the ear but extending to other portions of the organism as well and productive either of the partial deafness of childhood or of the somewhat indefinite state known as the "tendency" or "predisposition" to become deaf in later life.

The acquired conditions which result in deafness, on the other hand are more pathological in character, and they arise (1) from diseases of the ear, either primary or secondary, and (2) from injuries to the ear which are usually traumatic in origin.

Primary diseases of the ear are those which originate in the ear itself, and secondary diseases of the ear are those which result from general systemic diseases, such as the exanthemata and other infectious fevers and diseases, including syphilis and tuberculosis.

Inherited Deafness.

Time was when the existence of inherited deafness could reasonably be questioned, but that time has now passed. Recent investigations have shown conclusively that heredity is the great predominating factor in the causation of so-called congenital deafness, and there is good reason to suppose that much of that usually classified as acquired deafness is also of hereditary origin.

Dr. Edward Allen Fay, working in conjunction with the United States Bureau of the Census, and at the suggestion, I think, of Dr. Alexander Graham Bell, has been engaged for many years in the collection of data and the tabulation of statistics relating to the educationally deaf, and the result is a book by Dr. Fay, entitled "Marriages of the Deaf in America." This book is replete with carefully tabulated statistics based upon a personal correspondence with thousands of deaf persons whose names and addresses were furnished by the enumerators of the eleventh census, and I have summarized the conclusions as follows:

Marriages of the deaf are far more common in our country than in Europe, and during the past century they have increased enormously, their increase being proportionate and simultaneous with the increase of schools for the deaf.

Marriages of the deaf with the deaf are more numerous than those with hearing persons, and they are only a little less productive than ordinary marriages.

Marriages of the deaf are exceedingly liable to result in deaf offspring.

Marriages of the congenitally deaf are more liable to result in deaf offspring than are those of the adventitiously deaf

Deaf persons having deaf relatives, however they are married, and hearing persons having deaf relatives and married to the deaf, are very liable to have deaf offspring, the percentage of marriages resulting in deaf offspring and the percentage of deaf children born therefrom being almost invariably highest where both of the partners had deaf relatives and next where one of them had deaf relatives and the other had not, and least where neither had deaf relatives.

The marriages of the deaf most liable to result in deaf offspring are the consanguineous marriages. Out of 31 such marriages, 14 or 45.1 per cent. resulted in deaf offspring, and of the 100 children born from these marriages, 30, or 30 per cent. were deaf.

The investigations of which this is a summary have been confined hitherto to the congenitally deaf or to those acquiring deafness so early in life as to place them for all practical purposes in the congenital class, but I am informed that the plan is about to be enlarged so as to include in the future a study of all classes of deaf persons whatever may be the character or cause of the affection. This will be a great improvement, for it will serve to clear up some mooted points in connection with the etiology of what has been called sporadic and acquired deafness. I have long been of the opinion that heredity is an important factor in the causation of these forms of deafness, as it is in the educational form, although perhaps this theory may not be so easily proved.

The sporadic cases of congenital deafness are generally supposed to be due to such diseases and conditions as syphilis, tuberculosis, alcholism, insanity, idiocy, and the like, and in as much as these diseases and conditions have themselves been shown to be largely transmissible, I see no reason why the conditions giving rise to such forms of congenital deafness may not be regarded as having an hereditary origin. Moreover, by far the greater number of cases of so-called acquired deafness may be similarly explained. They are acquired because inherited physical anomalies have made their acquirement easy and in some cases perhaps unavoidable, and thus they may be regarded as indirectly inherited.

True hereditary deafness has been limited in our classifications to that of those showing a clear family history of deafness, either in direct or collateral branches, and this limitation has been made because of the mistake of supposing that deafness is the particular thing inherited, whereas, as I have said, what is really inherited is the physical anomalies which result in deafness. These physical anomalies may be transmitted by those having other forms of defectiveness in the same manner

as they are transmitted by those having a family history of deafness. Thus even the seemingly acquired deafness of adult life, or so-called catarrhal deafness, may be regarded as hereditary in so far as the particular physical and systemic conditions which cause it, such as rheumatism and gout, are hereditary or transmissible, and reasoning thus, we may be justified in the conclusion that all deafness with the exception of that of traumatic origin is in part at least due to some hereditary taint. Whether or not I am right in this supposition, time and further study will reveal, but we now know beyond any doubt that heredity is an important and increasing factor in that most serious and distressing of all forms of deafness, namely, the congenital form, and the question arises, what are we going to do about it? What prophylactic remedy is indicated?

The procedure that naturally suggests itself is embodied in the phrase, "a stricter application of the principles of eugenics" to the subject of deafness And what does this mean? It means, among other things, a more determined effort on our part to check the transmission from parents to offspring of the particular physical anomalies which result in deafness. And how can this be done? It can be done most effectually, of course, by rendering those who transmit such anomolies physically incapable of reproduction.

In a paper read before the American Academy of Medicine, in 1900, I advocated sterilization for the prevention of crime, pauperism, and mental deficiency, and I am pleased to note that the procedure is now being sanctioned by law and practiced in Indiana, California, Oregon, and Connecticut.

Moreover, there was recently appointed in the state of New York, a special committee on procreation, composed of three physicians, and their first meeting was held in Albany only a few months ago. We shall watch with interest the action of this committee, and it is a matter of congratulation, I think, that it should be composed of physicians who are in a position to know the facts which should warrant any action on the subject. Who knows but that the time may come when the

practice of sterilization, drastic as it now seems to us, will be adopted even for the prevention of hereditary deafness.

Another effective measure, and one less radical, would be the denial of the marriage license to those who transmit the physical anomalies which result in deafness. This measure should be considered at once. Surely the "strong arm of the state" should be used to regulate such marriages of the deaf as are known to result in a high percentage of deaf offspring, and this is especially true in view of the fact that the state is doing all that it can to make these marriages possible by bringing the deaf together in large institutions and increasing their economic efficiency. In other words, our efforts to ameliorate the conditions of the deaf have tended to foster their increase. In the first decade of the last century, the proportion of marriages of the deaf to the whole number of marriages during the century was only 0.02 per cent., and it increased with the increase of the number of schools during each subsequent decade until in the ninth, it was 22.7 per cent.

This fact suggests as another possible preventative measure, a change in our methods of education for the deaf. Instead of bringing them together in large residential schools, of which we now have about 150, with upwards of 13,000 pupils, I would recommend special day-school instruction, which would not only obviate the state of propinquity which is so likely to result in marriage, but it would have the additional advantage of keeping the deaf in closer association with hearing people during their educational career.

These are somewhat radical measures for the prevention of deafness, but they are measures which should be discussed and which sooner or later must receive serious consideration. We are face to face with conditions which have led close students of the subject, like Dr. Alexander Graham Bell, to fear the possible "formation of a deaf variety of the human species," and thus they seem to call for drastic measures. In the meantime, we can do much by a campaign of education in which the truth may be disseminated and the facts in the case given the widest possible publicity.

Those of us who know should discourage as much as possible the marriages of the deaf which are liable to result in deaf offspring. I use the word discourage because the times do not yet seem to be ripe for absolute legal prevention. We may, forsooth, control the breeding of our cattle, but our modern notions with reference to personal liberty, especially as it relates to the question of marriage, are such as to interfere very materially with the application of eugenic principles. I am a believer in "personal liberty," but when it interferes with the happiness and usefulness of unborn generations of fellow persons, I think it should be at least curtailed, and if necessary prohibited by legal measures.

Acquired Deafness.

When the measures which I have suggested for the prevention of inherited deafness shall have been more generally adopted, we shall have fewer of the conditions or anomalies which make the acquirement of deafness so easy and in some instances almost unavoidable. When we shall have eliminated heredity as a factor in the causation of deafness, the prevention of acquired deafness will be greatly facilitated. As it is, I am of the opinion that acquired deafness is preventable in the great majority of instances, and I am thus optimistic not because I have any new remedies to exploit, but because I have so much confidence in the old ones when applied in a timely and rightful manner.

The way to prevent acquired deafness is to prevent the development of those pathological conditions of which it is the result. Primary diseases of the ear proper are exceedingly rare. Most of the so-called primary ear diseases arise in the naso-pharynx, and their secondary effects upon the ear are largely preventable by a stricter application of well-known remedial principles.

Our modern surgical treatment of adenoids is probably the best procedure yet devised for the prevention of acquired deafness, but it could be made far more effective by doing the operation earlier and by making it more general and more thorough. Many ears are still being sacrificed to tardy and

careless surgery in this region. As someone has said, we are in the habit of calling the fire engine after the house has been burned, and I may add that the coming of the fire engine often renders the house uninhabitable. Our operations are performed in too many instances for the sole purpose of relieving some immediate symptom and with too little consideration for the saving of an important function. I have called attention to this point in connection with the tonsil operation. Just as the surgeon has not done his full duty in a tonsil operation until he has improved the speech and voice, so has he not done his full duty in other operations in this region until he has eliminated the acting and predisposing causes of deafness.

Moreover, as a further preventative measure, I would advocate a thorough and careful examination of the ears of very young children and an earlier treatment of the conditions which have been found to result in deafness This work should not be left entirely to the general practitioner, but it should be done in part at least by the skilled otologist.

Another important measure is that suggested by Dr. Sohier Bryant, namely, a periodic examination of all ears similar to that practiced with reference to the eyes, in order to detect and enable us to treat threatening conditions. Early examinations and treatment are especially indicated in those cases showing a family history of deafness.

The exanthemata and other infectious fevers of childhood are common causes of acquired deafness, and their prevention, of course, should be regarded as among the possibilities of the near future. Notwithstanding the attitude of certain foolish sentimentalists towards vaccination, smallpox has been almost entirely eliminated as a factor in the causation of deafness, and typhoid fever bids fair to have a similar fate. May we not hope that scarlet fever, measles, whooping cough, mumps, and cerebro-spinal meningitis may soon follow in their wake? In the meantime, no effort should be spared to mitigate the awful effects of these diseases upon the ears of children. This is a work that calls for a fuller coöperation of the general practitioner with the specialist, and it is no credit to either

that about 25 per cent. of all cases of acquired deafness should have their origin in these infectious diseases.

Interesting investigations are now being carried on across the water with reference to syphilis as an etiological factor in the causation of deafness. It has been shown that in the congenital form it may cause congenital deafness, and that it is an undoubted factor in the causation of acquired deafness. In view of these facts, Dr. James Kerr Love suggests as a preventative measure, making the pregnancies of syphilities and all still births notifiable, in order that preventative measures of treatment may be promptly adopted. Attention has also been called to the fact that our modern remedy, salvarsan, when given for the eye complications of congenital syphilis, has seemed to have the effect of actually impairing the hearing.

Finally, as has been well said, the prevention of deafness is largely the work of the Eugenist, the Hygienist, and the Legislator. It is the function of the eugenist to see that children are well born, of the hygienist to see that the mother is cared for during pregnancy and the child immediately and remotely thereafter, and it is the function of the legislator to enact such laws with reference to deafness as the eugenist and hygienist find to be desirable and practicable. Moreover, in these days of progressive medicine, the physician himself should be a eugenist as well as a hygienist, and he should take a leading part also in the adoption of the laws which make for health and happiness.

Discussion.

Dr. Max A. Goldstein, of St. Louis, said this interesting work of Bell and the valuable and voluminous statistics paper, with the series of deductions from the well-known from Mr. Fay concerning the marriages of the deaf, will admit of more discussion and consideration before the initiation of the drastic measures recommended. As a matter of fact, many who have examined large numbers of children in schools for the deaf know that much which is ascribed to inherited deafness is not inherited deafness at

all. Many of these children have acquired deafness on account of some febrile condition when under two years of age. Many of the conditions given by parents who are more or less ignorant about the causes of deafness are erroneous. "Brain fever" may make of an absolutely normal child a deaf subject. There should be a much more careful consideration of the causes of deafness and more careful examination. Many cases now in the category of inherited deafness may be placed in that of acquired deafness. There are doubtless many more cases acquired from scarlet fever and other exanthemata than are inherited deafness. If that is true, pupils who have acquired deafness should not be subjected to such drastic measures as those recommended for the prevention of deafness. A child brought up in a school for the deaf who has acquired deafness may not transmit it more than any other subject. He had been trying to get a little closer to the proposition with reference to Catholics. In institutions for teaching pure oralism the statement has frequently been made to him that there should be as many Jews and Irish as possible. That is simply borne out by experiment and may be in support of Dr. Makuen's problem. The same classification which Bell makes in his "Transmission of Inherited Traits," is also made by Alfred Graves of Edinburgh with reference to otosclerosis. Of course otosclerosis is an inherited condition, and it is the pure congenital type of cases that appearances may be carried out.

Dr. Sargent F. Snow, of Syracuse, N. Y., thought it well enough to add a word of caution with reference to intermarriage. The question of eugenics is not so far-fetched as some seem to think. All who know about the breeding of animals know how a type can be developed at will and how types of animals can be fixed by in-breeding. It would not be stretching the point to ask legislative action which would prevent the intermarriage of people who have a strong tendency to deafness. A law could be so framed as to prevent such intermarriage of blood relatives.

Dr. Makuen replying to Dr. Goldstein said that he would emphasize the importance of keeping in mind that it is not deafness that is inherited, but it is the physical anomalies which give rise to deafness and thus many of the cases of so-called acquired deafness really come under the inherited class. On cannot read Dr. Fay's book carefully without being convinced that inheritance is probably the most important factor in the causation of deafness, and Dr. Bell's chart, which I have shown you, also emphasizes this point. Here is a man who became deaf as a result of "brain fever," and in three generations of descendants there were 17 deaf born children or children who became deaf soon after birth. It makes no practical difference whether a child is born deaf or whether he becomes deaf during his first year, and yet the one is called a congenital case and the other an acquired case. The fact is that both probably inherit physical anomalies which result in deafness, one a little before and the other a little after birth.

REPORT OF A CASE OF TEMPERO-SPHENOIDAL ABSCESS OF OTITIC ORIGIN. OPERATION. RECOVERY.

By JAMES F. McCAW, M. D., Watertown, N. Y.

The subject of this report was that of a female 44 years of age, married, seen in consultation April 27, 1912, at the City Hospital. The following history was obtained: She had suffered from a severe head cold six weeks before, which was followed in a few days by severe pain in the right ear and discharge thirty-six hours later, with only slight relief of the pain; these symptoms continued, and for about three weeks before coming to the hospital, she had suffered such severe pain over the entire right side of head that the physician in attendance had kept her under the influence of large doses of morphine and free administration of whisky. On the above date another physician was called and, realizing the gravity of her symptoms, sent her at once to the hospital and asked me to see the case. Upon examination her temperature was 100.6 F., Pulse 102, Respirations 20; her face showed great suffering and anxiety, there was a very profuse thick creamy purulent discharge from the right Auditory Canal, the Membrana Tympani was intensely congested, thickened and œdematous, there was a small oval perforation in the lower posterior quadrant through which pulsating pus was being discharged. There was such marked bulging and drooping of the upper posterior quadrant and the posterior superior canal wall that the perforation was just visible. There was decided swelling and oedema over the entire mastoid area and extending well above the antral region. This entire area was exquisitively sensitive to touch, and severe pain elicited from the slightest manipulation. On assuming the erect posture in bed, dizziness was complained of, mentality seemed dull, but all questions were answered correctly, no nausea or vomiting. Convulsive

seizures were absent and no evidence of muscular paralysis. Both eye grounds were normal. Urine analysis negative. I operated upon this case the next morning, using the usual Mastoid incision. After elevating the flaps and periostium over the mastoid area, and also from the tip, a very extensive bone necrosis was encountered so that the lower half of the external table, including the tip, was a sequestrum and was lifted out whole. From necrosis of the inner table, about an inch and a half of the Sigmoid Sinus was exposed, covered with pus and exuberant granulations. After cleaning out all necrosis and granulations, my attention was next given to the tegmen Antri, which was soft, and with a curette and rongeur this was carefully cut away exposing the underlying dura, which was very much thickened, discolored, and felt resistant. An incision through the Dura at this point opened directly into an abscess cavity from which was evacuated about a dram and a half of pus. The cavity, which was about 1½ inches in depth, was carefully wiped out with cotton sponges, and upon examination there was no apparent limiting membrane, but the cavity seemed to be directly within the brain tissue clearly demonstrating that it was a very acute and recent process. The outer opening through the Dura was enlarged and a strip of Iodoform gauze was carried in and packed loosely into the cavity. The Mastoid wound was dressed in the usual way.

Blood count at this time showed a Leucocytosis of 13,200 and a Polynucleosis of 84%. For three days following the operation the patient complained of considerable and severe pain over the right side of head, with nausea and vomiting, restlessness and irrational periods. The temperature kept steady between 100F. to 100.8F. during this time, and pulse from 78 to 90. Blood count taken each day showed diminishing absorption with good resistance, so we began to look about for something else to account for this train of untoward symptoms which seemed quite alarming, when it occurred to the writer that perhaps the withdrawal of the amount of whiskey and morphine which she had been accustomed to for six weeks was responsible for this post-operative development. With

this idea she was put on Chloral gr III and Sodium Bromide grs XV three times daily and at night if necessary.

It was a most noticeable fact that all the Cerebral symptoms disappeared promptly, her temperature was normal two days later, and she had no further symptoms of this character. The first dressing was done on the fifth day, and the gauze removed from the abscess cavity, which was perfectly clean and without retention, the dural opening patulous. The cavity was dried out with pledgets of sterile cotton and one strip of Iodoform gauze inserted. It was dressed every second day, and on the ninth day following the operation all gauze was left out of the abscess cavity, as it had contracted down to almost nothing and simply showed a depressed granulating surface. From this time on the patient made an uneventful recovery. This report is made not so much for its importance as a contribution to this society, but simply that it may be on record for its clinical value. However, there are several points of interest in it from a clinical standpoint. The first to be noted is the difficulty in making a correct diagnosis of intracranial complications before operation. This case illustrates this point very clearly as the patient gave no symptoms of brain abscess prior to its discovery at the time of operation, the dizziness complained of, I think, can be explained by the extreme intratympanic pressure, the mental dullness was attributed to the influence of the morphine which she had been taking.

We also have in this case an illustration of the importance of weighing very carefully all post-operative symptoms and eliminating all possible sources of their development before deciding that they are dependent upon an extension of the original infection, and still further complicating the case by an unnecessary secondary operation. I feel quite sure that had I assumed from the misleading post-operative symptoms present that there was further brain involvement, and made an exploratory operation I would have jeopardized my patient's chance of recovery.

I want to again call attention to the value of daily blood counts as an aid in arriving at a correct diagnosis in cases when there is much apprehension and puzzling symptoms.

Especially is it of importance in post-operative cases of septic intracranial involvement which may, as in this case, develop alarming symptoms dependent upon some intercurrent condition. It was of the greatest value to me in this particular case, as I would have been strongly inclined to re-open the brain wound in view of the alarming symptoms had not the blood counts taken daily shown a diminishing Polynucluosis and a steady Leucocytosis, thus shortening the resistance line, which has been pointed out by Sondern to be a good indication.

My method of packing the abscess cavity might perhaps be criticized in view of the experience of many of our best operators, but my decision to use the Iodoform gauze was based upon the fact that it was a small cavity showing a tendency to contract, and that I was able to enter it directly along the route of infection, therefore in all probability the brain coverings were thoroughly amalgamated over this area, thus minimizing the possibility of a septic meningitis. The result in this case was a perfect recovery.

Discussion.

Dr. Edward B. Dench, of New York City, thought it a mistake to renew the dressings too quickly. In the case of an acute abscess all manipulations injured the delicate membrane. By simply opening the abscess and draining the best results could be obtained. It had been his custom lately to open the abscess, put in a cigarette drain, and trust to nature to accomplish the rest. It should be remembered that in all acute cases the patient is suffering not only from the presence of pus but from cerebritis, which will be aggravated by manipulation in an endeavor to drain every nook and corner of the abscess.

Dr. B. Alexander Randall, of Philadelphia, referred to re-reading the observations of Sir William Macewen, who advocated opening these abscesses and curetting them just as he would a surgical wound anywhere else and reported seventy-five to eighty per cent. of successes. The speaker had

formerly opened the abscesses and left them alone, and had had success in from thirty to forty per cent. of cases, whether because of or in spite of his method, he did not know. He would like to know Macewen's later results; he did not believe he had kept up the former record.

Dr. John F. Barnhill, of Indianapolis, Ind., asked what the symptoms were which led to the opening of the dura. One should be very explicit in describing such cases. The case reported was an acute abscess without abscess sac. There was authority on both sides with reference to the removal of the sac. All who have tried to remove it had found it vastly more difficult to accomplish than to leave it alone. It had always seemed to him to act like nature's best drainage tube; to disturb it would be bad surgery, just as it would be to disturb adhesions around an old infected appendix. He had had good results in cases in which he had left the sac.

Dr. Foster Kennedy, of New York City, came from the neurological rather than the otological point of view to hear something of the method of diagnosis which was adopted in this case. According to the observations of great surgeons, in cases in which there is a history of old middle ear disease, in which the bone has been eroded, and in which the temporo-sphenoidal lobe is involved, it is difficult to tell, whether there is a tumor or an abscess in the temporo-sphenoidal lobe. There are certain things which make it comparatively easy, if the symptoms are looked for, to say that this locality is diseased. From lesions of the temporo-sphenoidal lobe come the curious, reminiscent, dreamy states described by Hughlings Jackson several years ago. These dreamy states are characteristic of temporo-sphenoidal lobe disease on either side of the brain. The result of pressure on motor areas from this lobe is hemiplegia of the face out of all proportion to that of the leg and arm. The reason for this is that the temporo-sphenoidal lobe lies immediately below the fissure of Sylvious, above

which is the face center. Pressure striking upwards and inwards thus affects the face out of all proportion to the arm and leg. Going inwards it strikes the optic thalamus, which controls muscular emotional expression. When such a person laughs he is able to express his emotion with the ipsolated but not the contralateral side of the face. The power of expressing emotion with the affected face is limited. That is frequently seen in tumors of the temporo-sphenoidal lobe. Aphasia is another symptom. The type of aphasia, according to textbooks, is for word deafness; that is not the case. The center in which words are stored is in the transverse gyri of Heschl. Without complete destruction of these gyri there is no word deafness. The result of graduated injury to that center is deterioration of memory for names. The characteristic things in this aphasia is that patients are unable to remember names of things observed. He cited the case of a stenographer who had this form of aphasia. When shown a fountain pen the patient called it "one of those patent pens." Operation was performed on the basis of that symptom alone, and an enormous tumor of the left temporo-sphenoidal lobe was found. The prognosis is graver with chronic abscess than with tumor of the temporo-sphenoidal lobe. He agreed with Dr. Barnhill with reference to removal of the entire abscess. If it is possible to do this it is the operation of choice. But it makes a difference whether the abscess is on the right or left side of the brain. If on the right side, remove it. He had seen a tumor weighing 120 grams removed from the temporo-sphenoidal lobe without any symptoms afterward. The left temporo-sphenoidal lobe is the most vulnerable portion of the brain. Through it pass all the association tracts connected with communication with the external world. If the abscess is in the right temporo-sphenoidal lobe remove it entirely; if it is in the left, open, drain, and leave the brain as nearly intact as possible.

Dr. Emil Amberg, of Detroit, Mich., asked Dr. McCaw whether he regarded the change mentioned as characteristic

of brain abscess or brain tumor; also whether it could exist without either abscess or tumor. The speaker believed this possible. He emphasized the multiformity of brain abscesses and the consequent difficulty of comparing one with another. He doubted that a recovery of 60 or 75 per cent. of brain abscesses could be duplicated. We might come nearer to the understanding of real conditions if also all the cases would be reported which have been operated upon without success.

Dr. Eugene A. Crockett, of Boston, Mass., reported a case similar to that detailed by Dr. McCaw, in which the man had a discharge of a few drops of pus on Monday, with a temperature of 102° F., and a pulse of 160. The family physician asked on Thursday if this should give alarm, to which the speaker replied in the affirmative. Examination showed the Babinski sign to be present; reflexes otherwise normal. Paracentesis was done, and a large quantity of clear pus evacuated. About five o'clock the next morning the patient's sister, who was taking care of him, found that he had something the matter with his speech. When he saw the man at seven o'clock there was no edema, and the reflexes were normal, but he had typical ephasia, with loss of ability to name any object. He was operated upon on the fifth day, a temporo-sphenoidal abscess being found. About a teaspoonful of pus of the strepticoccus variety was evacuated. Lumbar puncture gave negative culture. That afternoon and next day the aphasia completely disappeared, but the patient died of septic meningitis three days later. In this case there was a completely encapsulated temporo-sphenoidal abscess on the fifth day after the onset, with clear fluid by lumbar puncture.

Dr. S. MacCuen Smith, of Philadelphia, said Dr. Kennedy's remarks suggested to him the point he had brought out in a recent paper concerning the indescribable facial expression of the patient in these cases. This facial expression, together with the aphasia, seemed to warrant the decision to operate for temporo-sphenoidal abscess. He

endorsed the statements and the findings of Dr. Randall and Dr. Dench. There should be the least possible interference, in evacuating a brain abscess cavity. To proceed as in the case of an abscess cavity in general surgery would be a mistake.

Dr. McCaw, in closing the discussion, said, in reply to Dr. Barnhill's question with reference to diagnosis, that he was prompted to open the dura by the fact that, after cleaning out the mastoid, he found the tegmen antri softened, the dura thickened and discolored at that point. In the presence of a mastoid necrosis of six weeks standing with the above findings in reference to the dura, was sufficient evidence to warrant exploration. He was glad to see that men of such wide experience as Dr. Randall and Dr. Dench agreed with him in this line of treatment.

A SKULL TREPHINED FOR MASTOID CARIES AND LATERAL SINUS THROMBOSIS BY THE LATE DR. SAMUEL ASHHURST OF PHILADELPHIA SOME FORTY YEARS AGO.

By B. ALEXANDER RANDALL, M. D., Philadelphia, Pa.

The specimen which I bring to your notice was given to me by my friend and colleague, the late Dr. Samuel Ashhurst, a dozen years ago, as obtained from a patient upon whom he had operated at the Episcopal Hospital in Philadelphia some thirty years before. Its importance did not at once appeal to

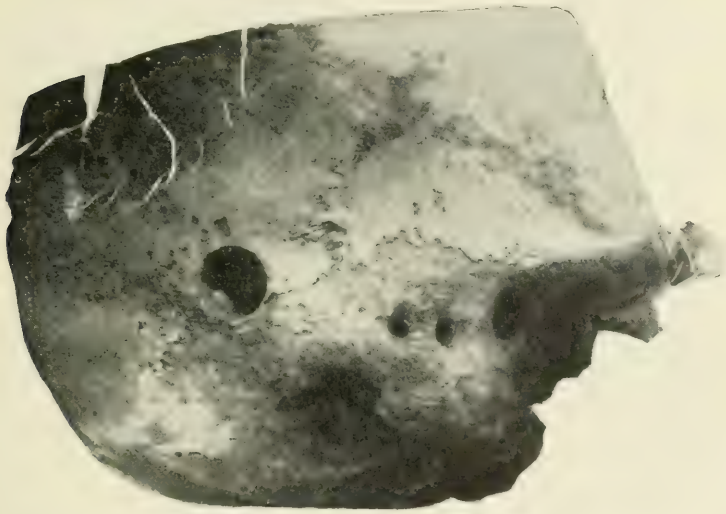


Fig. 1. Exterior showing two mastoid openings eroded like the surrounding surface and the trephine opening over the sinus.

me, preoccupied as I was at the time by concern for diphtheritic conjunctivitis with which he was suffering, and I laid it aside without obtaining from him before his death a little later (Nov. 12, 1901), an explicit account of the case, with name

and dates. Now when I try to locate it among the Records of the Hospital, I find that they are incomplete for that period. As there is a quite full record of operations since 1875, however, the negative search at least corroborates the verbal statement that the operation was done some forty years ago. In default of the history-notes I am unable to give any details as to the symptoms and course of the case; but must let the specimen speak for itself.

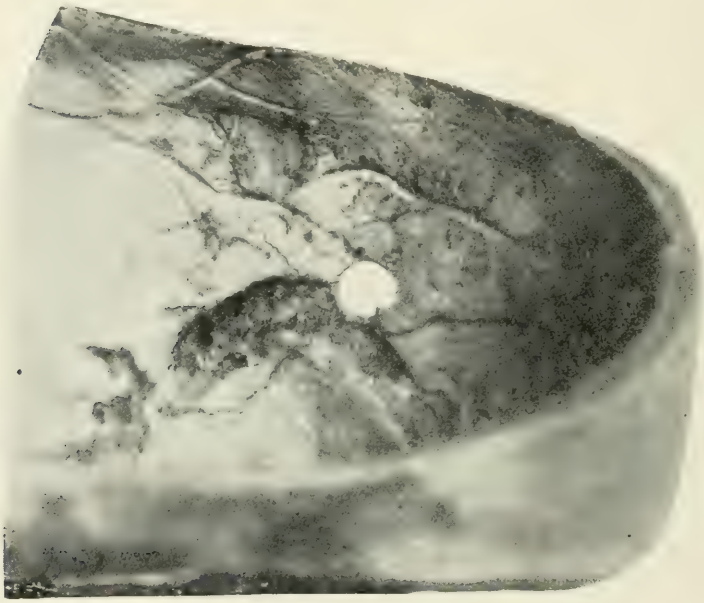


Fig. 2. Inner aspect showing curious mastoid, eroded sulous and the trephine opening giving access to it.

It is the posterior half of a rather massive skull without calvaria, the nearly transverse section passing through the middle of the *sella turcica* and zygomata. The left temporal bone presents no lesion—the drum-membrane is gone but the ossicles seem normal. On the right the malleus is in place, but the drumhead, incus and stapes are lacking, unless it be the foot-plate which closes the oval window. No caries is ap-

parent in the tympanum. The mastoid shows erosion of much of its external surface, as do two nearly circular openings some five mm. in diameter, which seem to have been made with a conical drill. If artificially made, these must date from some days or weeks before death. The anterior opening, 8 mm. down and back of the *spina supra meatum,* enters the carious cavity of the mastoid; the posterior, centered on Reed's base line 16 mm. back of the spina, enters the sulcus of the sigmoid. Centered 45 mm. back of the spina and 10 mm. above the base line a half-inch crown-trephine has been used and a button of the parietal removed. The sharp cut edges would indicate that this intervention had but slightly antedated the patient's death.

Study of the internal surface shows two perforations of the upper surface of the petrous, 2 mm. and 4 mm. respectively in diameter, which communicate with the carious mastoid cavity, near the junction of the superior petrosal and lateral sinuses; but little erosion in the middle cerebral fossa. strongly hollowed sulcus of the lateral sinus is eroded from near the torcular forward almost to the jugular bulb and at the knee communicates by a rough opening 15 mm. in diameter with the carious mastoid. The trephine-opening is at the upper margin of the sulcus—the posterior drill-hole at its most anterior part; both should have given access to the peri-sinous abscess.

We have evidence here of middle-ear suppuration, probably chronic, opening back into the sulcus and leading to repeated interventions to drain the mastoid, free the sulcus of its pus and then, upon just what indications we may only guess, making a formal trephining for freer access to the lateral sinus—and this done about a decade before Zaufal, Horsley, Lane or Ballance had taken up the subject.

THE PATHOLOGY OF THE LABYRINTH AS SHOWN IN MICROSCOPIC SECTIONS (STEREOPTICON DEMONSTRATION).

By MAX A. GOLDSTEIN, M. D., St. Louis, Mo.

The study of the physiology and pathology of the labyrinth, which has received such a marked impetus in the past five years, especially through the achievements of the Vienna school, has stimulated much thought and activity along these lines in America. Unfortunately, however, our clinical endeavors overshadow our pathological activities, and, as yet, there has been comparatively little work done in America in the pathology of the labyrinth.

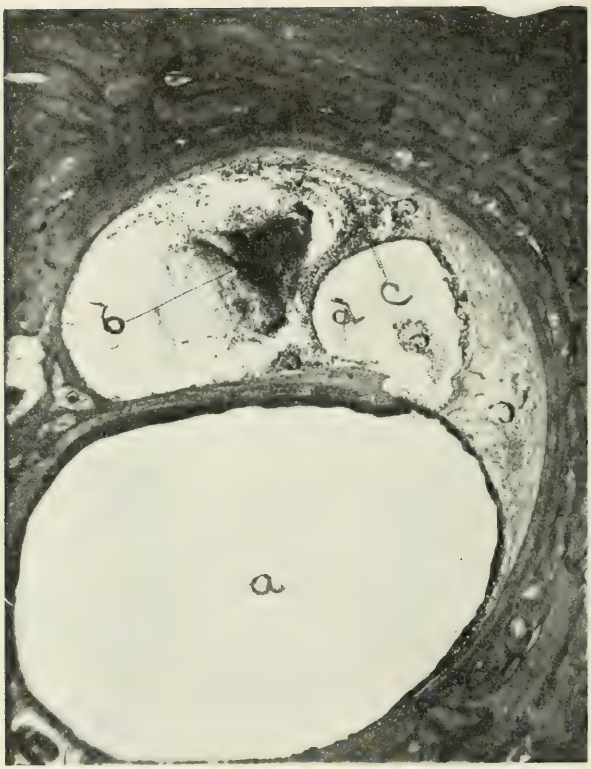
This is due largely to the fact that the busy otologist has little leisure, partly to insufficient laboratory training, but especially to the stringent regulations in our country, which prevent prompt and unrestricted post-mortem examinations.

Post-mortem examinations on the labyrinth must be undertaken within a few hours after death if we wish to make a careful study of the histo-pathology of this area, for the delicate structures of the membranous labyrinth are very perishable and soon show tissue-changes and artefacts if precautions for their immediate conservation are not taken.

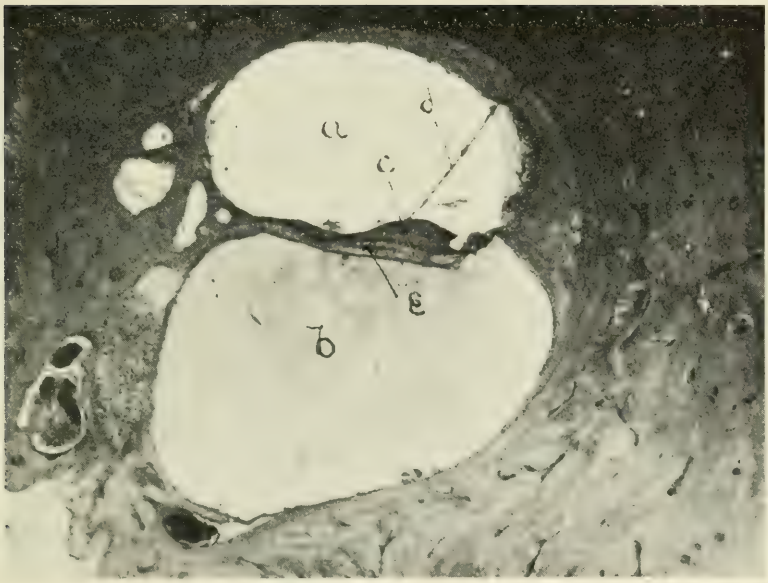
All this serves to point out briefly the difficulty of obtaining satisfactory pathological material for the active study of the microscopic normal and pathological labyrinth.

In presenting this small series of slides illustrating the more common types of labyrinthine pathology, I do not claim much originality, but hope to stimulate our Fellows to more active work in this resourceful field of research.

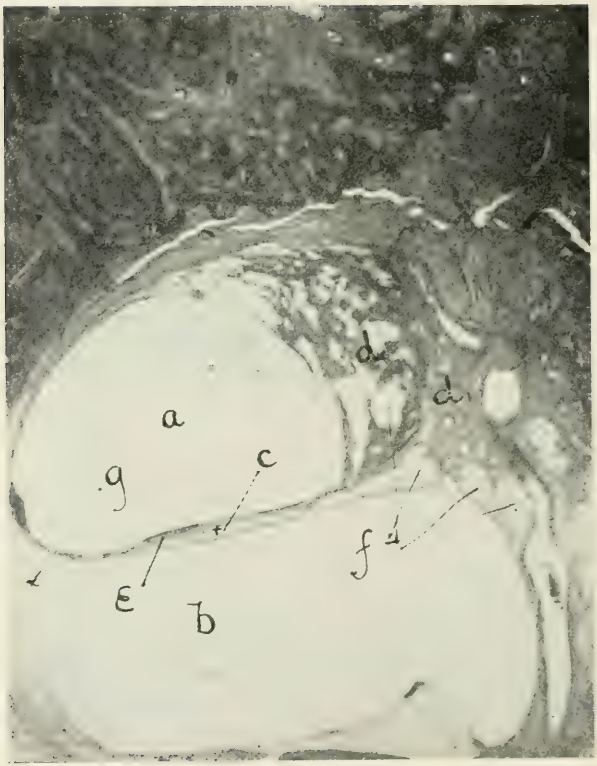
1. Congenital Deaf-Mutism. Cochlea with total absence of nerve element. Ectasia of ductus cochlearis. Distended member. Reisnerii. Dropsic degeneration of ligamentum spirale. (a) Scala vestibuli; (b) Scala tympani; (c) Lamina spirali; (d) Membrana Reisnerii; (e) Lamina basilaris; (f) Area of nerve sheaths and nerve distribution, showing absence of nerve tissue.



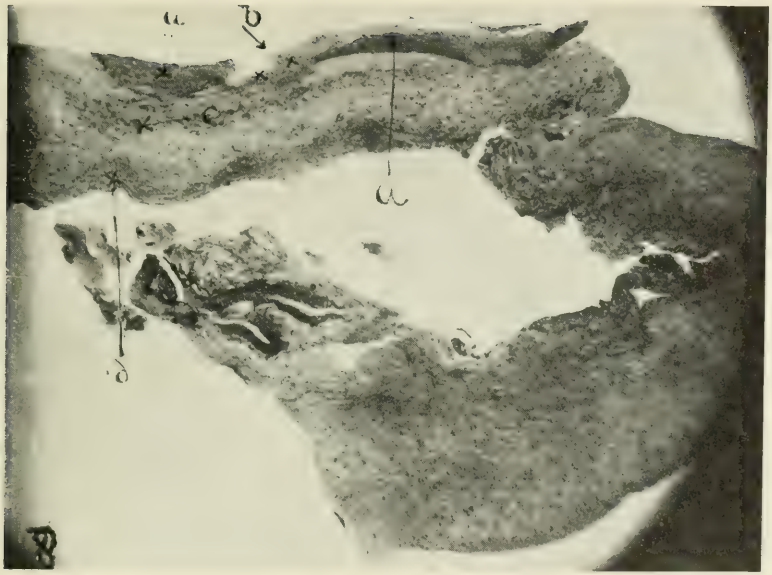
II Purulent Exudation in Cochlea, especially in scala vestibula of basilar turn. Pigmentation result of old hemorrhage in exudation, indicative of chronic suppuration. (a) Scala tympani; (b) Pigmentation result of old hemorrhagic infiltration; (c) Fibrinous shreds result of chronic suppuration; (d) Scala vestibuli.



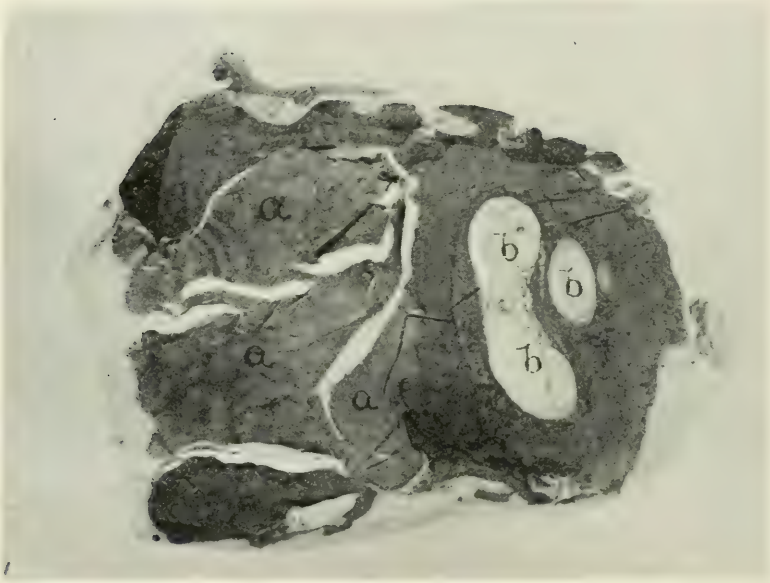
III. Serous Labyrinthitis. Infiltration throughout cochlea, but no destruction of minute membranous parts. (a) Scala vestibuli; (b) Scala tympani; (c) Membrana tectori; (d) Membrana Reisnerii; (e) Lamina basilaris with histological elements intact.



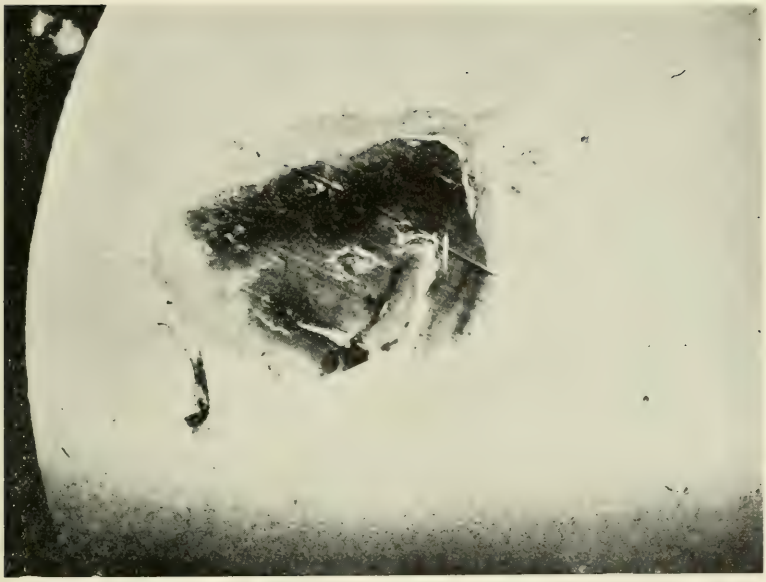
IV. Congenital Deafness. New born. No nerve tissue. New bone. (a) Scala vestibuli; (b) Scala tympani; (c) Lamina basilaris with absence of histological elements; (d) Ganglion spirale; (e) Organi Corti; (f) Areas of nerve cells minus histological elements; (g) Ligamentum spirale.



V. Purulent Thrombus in Wall of Jugular Vein. (a) Organized thrombus; (b) Broken down area of thrombus; (c) Wall of jugular vein.

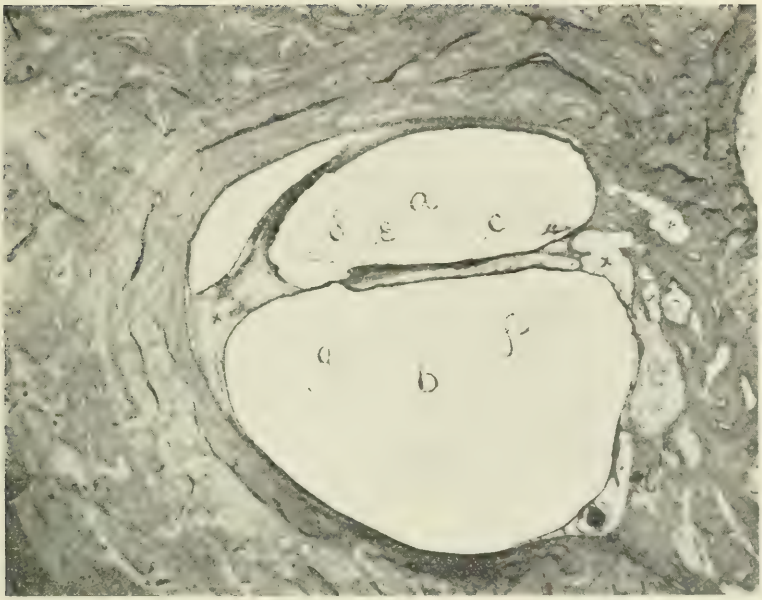


VI. Neuro-fibroma of Auditory Nerve. (a) Neuro-fibroma filling area of degenerated cochlear nerve; (b) Median section of cochlea.

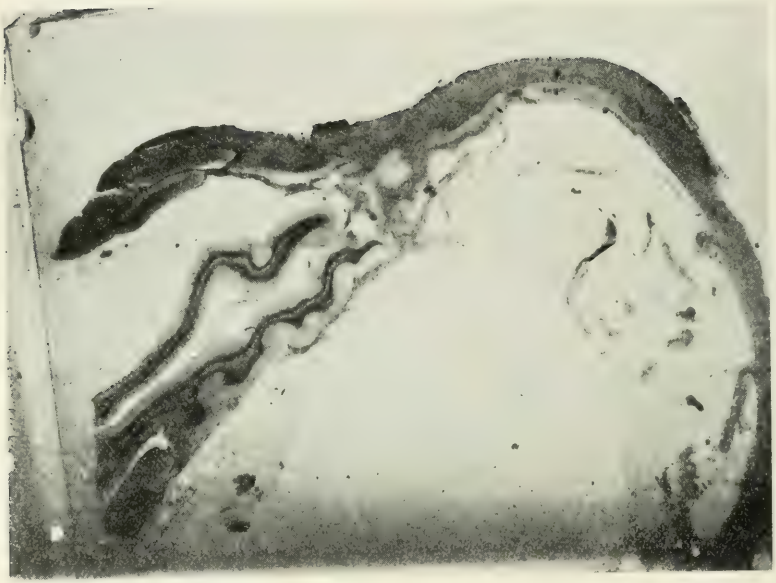


VII. Phlebitis of Lateral Sinus with Non-infected Thrombus

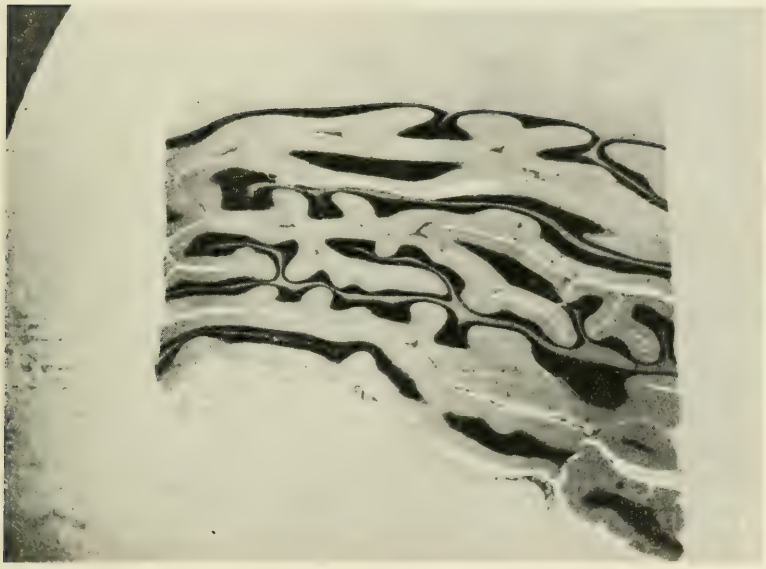
VIII. Deaf-Mutism. (Low power). Psamon—calcified albuminous kernels, usually found in wall of dura in internal auditory canal.



IX. Deaf-Mutism. (High power). Psamon—calcified albuminous kernels, usually found in wall of dura in internal auditory canal. (a) Scala vestibuli; (b) Scala tympani; (c) Lamina spiralis with defective histological elements; (d) Degenerated membrana Reisnerii; (e) Membrana tectori; (f) Defective nerve ganglia; (g) Ligamentum spirale.



X. Brain Abscess Wall. No line of sharp demarcation between abscess wall and meninges.



XI. Meningitis

REPORT OF CASE OF SUPPURATIVE LABYRINTHITIS. RECOVERY WITHOUT OPERATION. COMMENTS ON DIAGNOSIS AND INDICATIONS FOR TREATMENT.

By O. A. M. McKIMMIE, M. D., Washington, D. C.

The following brief case report is presented with two objects in view; first because I believe that every case of labyrinthine disease recorded furnishes additional data for determining more accurate indications for treatment; and second because the cases in which healing occurs without operation tend to make us look more conservatively upon the labyrinth operation, which in the minds of many of us is not without danger per se.

In March, 1902, Mrs. C. had an abscess in the left ear following what was apparently an ordinary severe head cold. In June of the same year she came under my care with a chronic suppuration, the ossicles being apparently intact although the lower half of the drum head was absent. Treatment for about one month improved this condition but did not cure it.

She then passed out of my hands for some time, and when seen at various times during the succeeding two years showed a progressive destruction of the ossicles and remains of the drum membrane.

The succeeding eight years she was out of Washington but her otorrhoea was more or less continuous and her symptoms such that the radical mastoid operation was under consideration several times.

In May, 1912, she came under my care again after several nights of severe pain. I found the middle ear filled with masses of granulations which blocked the attic and obstructed drainage. Appropriate treatment relieved her pain and unblocked the attic.

A little later I used a bead of fused nitrate of silver on the remaining and largest granulation mass, which grew from the tube mouth and the tymanic ring upward and backward.

The slough, thus produced, was removed two days later; and in taking it away a small necrotic bit of the tympanic ring was removed. The use of pure alcohol for about two weeks was followed by complete closure and epithelialization of the tube mouth; but some discharge from the attic still persisted.

The patient left the city July 15th, 1912, for the summer, and during September had a period of five days during which she suffered pain and intense and continuous vertigo.

She gradually recovered from this, and when I saw her again in the latter part of the month manifested no spontaneous symptoms of disturbance of equilibrium, but she still had a considerable discharge of thin pus from the ear.

For several years this ear has been profoundly deaf, and she had had occasional transient tinnitus. Investigation showed a totally deaf ear and there was no response to rotation, the caloric test nor to the test for fistula.

At this time all vestiges of the ossicles and membrana vibrans had disappeared and the opening into the attic was small. There was still a very considerable discharge, and I was strongly tempted to do a radical operation, but decided to first try routine cleansing treatment.

I was much surprised to find that, after getting the internal wall of the cavity perfectly dry, a shining spot appeared within half a minute on the promontory which when gently mopped gave evidence of a thin brownish purulent discharge.

Here was undoubtedly a fistula and the entrance point of the labyrinthine invasion.

As all spontaneous symptoms of internal ear disease had disappeared I believed that nature had filled the destroyed internal ear with protective granulations and that underneath the minute opening which I could see in the membrane covering the promontory there was probably a small area of necrosis.

Acting on this belief no attempt was made to enlarge in any way the fistula: and ordinary routine cleansing was carried out until October 8th, when she again left the city, returning November 1st.

From this date on the patient was seen daily and a weak hydrochloric acid and pepsin solution used and allowed to remain in the ear twenty minutes.

After ten days of this treatment the discharge began to lessen and about the middle of December ceased.

Today the patient is in perfect health with a dry ear; and the site of the perforation in the promontory wall is not visible. Its location, however, can be easily shown by pressure with a fine cotton tipped applicator which shows a momentary depression when the pressure is applied to the correct point.

Here is a case corresponding to Neuman's sixth classification, having hearing, labyrinthine function and fistula tests all negative and accordingly the radical mastoid and labyrinthine operation are indicated, if we accept his table of indications.

On the other hand a fistula is actually demonstratable and inasmuch as pressure or suction brought to bear upon it does not produce any evidence of excitability of the labrinth we are sure that the labyrinth is totally destroyed.

The question here arises as to the character of the lesion which has destroyed the vestibular structures and cochlea. With a purulent discharge coming from the fistula in the promontory wall it seems hardly reasonable to think that the whole process can be other than suppurative, inasmuch as a coagulative serous labyrinthitis, while giving a negative response to rotation and caloric tests, would in most cases give a positive response, under such conditions as exist here, to suction and compression. A positive response to the fistula test would show that some part, at least, of the vestibular apparatus is excitable, and so long as a positive response is obtainable operation on the labyrinth should be postponed

unless other conditions indicate probable or beginning extension to the cranial contents.

Furthermore in cases presenting a history of previous labyrinthine disturbance but having no symptoms of brain or meningeal involvement at the time of examination, and with negative responses to cochlear, vestibular and fistula tests I believe it safe to assume that nature has walled off the infective process and that the discovery of a fistula under such conditions is not an indication for operation. When in the course of radical operation a previously unsuspected fistula is discovered this in itself should not be taken as an indication for immediate destruction of the labyrinth.

When a radical operation is planned the patient should be carefully examined not only as to hearing, but also as to the excitability of the vestibular apparatus, and particular attention paid to the fistula test.

While the presence or absence of a fistula should not per se indicate or contra-indicate an operation on the labyrinth, a positive response to this test should cause us to hesitate about opening up widely a labyrinth which has a chance of recovery of partial function, unless other symptoms demand such a course: and further finding a fistula in cases such as here reported, in which the suppurative process has ceased, except at the fistulous opening, or in which it is evidently walled off from the cranial cavity a complete operation on the labyrinth is not demanded.

It is my belief that the case reported is one of chronic diffuse suppurative inflammation in which until the vestiginous attack there had remained some functionating power in the vestibular structures, and that the paths of possible extension to the cranial contents had been previously walled off, the vertiginous attack being the last cry of a dying vestibular apparatus: in other words, a gradual diffuse destructive process winding up with a sudden acute exacerbation. The practical lesson to be learned is, that in the presence of a non-reacting labyrinth—with or without a fistula—but with a chronic suppuration of the middle ear we may be in doubt as to whether we are dealing with a labyrinthitis which

is merely quiescent or with one which is practically healed. Experience has shown the performance of the radical mastoid operation frequently starts up a fresh inflammatory condition in the vestibular apparatus, and unless other patent symptoms demand it no operation should be done without first subjecting the patient to very thorough routine treatment for the cure of the suppuration: and if a radical is done, that is all that should be done at the first sitting, the patient being carefully watched for symptoms of beginning brain involvement such as sleeplessness, headache or beginning rigidity of the neck.

With our gradually increasing knowledge of labyrinthine infections the radical operation imposes a much graver responsibility on the surgeon than ever before.

Discussion.

Dr. Edward B. Dench, of New York City, thought there was a tendency to be a little less anxious to perform the radical labyrinth operation than when it was first described by Neumann and others. It should not be the procedure of choice. He cited a case in which there was a fistula. All labyrinthine tests had been made, the static labyrinth responded to the fistula test, the fistula was found, it was curetted, and, with a pair of forceps, a coagulum of pus was lifted out. That was all that was done to the labyrinth. The cavity was skin-grafted, and the patient made a complete recovery. It was different when the labyrinth was dead. In the case reported by Dr. McKimmie it would seem that the labyrinth would be better removed than left in place.

Dr. B. Alexander Randall, of Philadelphia, agreed with Dr. Dench that only what is absolutely necessary should be done in such cases. He wished, at the risk of being *malapropos,* to lay stress upon the futility of the "fistula symptom." He had seen it most marked in cases in which there had been no suppuration whatever, and where there was

only undue freedom in the oval window. He merely wished to impress upon the younger members that the fistula sign may be present when there is no fistula.

Dr. Ewing W. Day, of Pittsburg, Pa., said one isolated case is not a criterion by which to be governed in the majority of cases. He cited a case in which there had been chronic otitis media for years. The patient was sent to the hospital four weeks after an attack, lasting a week, of vertigo, nausea and vomiting. The caloric reaction and fistula test present. Hearing for high C present. Proceeding on the principle that if there is any hearing left operation is not indicated, the man was put to bed, with an improvement for four or five days. The cerebrospinal fluid was negative. A sudden exacerbation occurred, meningitis developed, and the patient died. At autopsy especial attention was paid to the internal ear, and it was found that the semicircular canal and the vestibule were filled with pus. The cochlea was as nearly normal as could be determined upon microscopic examination. If he had operated he would have had a better chance of saving the man's life than he had by the course he followed. On the other hand, in Dr. McKimmie's case there was oozing of pus, it was left alone, and the patient got well. One case could not be taken as a criterion for the other. A large number of cases stand a better chance of getting well if bone is removed and freer drainage given.

Dr. Max A. Goldstein, of St. Louis, Mo., agreed that too much stress cannot be laid upon the fistula sign. It should be borne in mind that there are two types of fistula sign: one is the fistula test, the other is the fistula sign which is brought about by ocular examination. Might there not be a small area of caries over the promontory and semicircular canal that does not extend into the membranous labyrinth? This might be mistaken for a destroyed bony labyrinth, accepted as corroborative evidence, and operation resorted to as a consequence.

Dr. McKimmie, in closing the discussion, thought Dr. Day's case very interesting. If he had seen his patient in September he would probably have operated. She came a few weeks later, when she had no symptoms of labyrinthine trouble; otherwise, having had her under observation for ten years with this chronic suppuration, he would undoubtedly have gone into the labyrinth. At present everything is healed perfectly, she is well, the ear is dry, and the labyrinth dead. The patient when seen the day before the reading of this report showed the "compensation symptom" when the right ear was subjected to the rotation test. Every case was a law in itself, and each must be handled according to its own requirements.

REPORT OF TWO CASES OF CEREBROSPINAL MENINGITIS PRESENTING NO CENTRAL NERVOUS PHENOMENA UNTIL SHORTLY BEFORE DEATH.

By T. PASSMORE BERENS, M. D., New York, N. Y.

Mr. X., age 59, consulted me January 10th, 1913. He complained of deafness in both ears and of pain in the right ear for a week, following influenza from which he was convalescing. Examination of the ears revealed impacted cerumen, the removal of which restored his hearing to such an extent that he heard a whisper at 3 ft. in the right ear and at 8 ft. in the left ear. The right membrana tympani was slightly reddened. He complained of headache, also of pain in the region of the right mastoid. There was no bulging of the membrana tympani, which was slightly injected, no oedema and no pain on deep pressure The patient had two uncomfortable nights, then returned because of pain on January 12th. The temperature was 97½°, the redness of the membrana tympani persisted. There was no bulging, no tenderness, but slight mucorpulent discharge from the accessory sinuses of the nose. His general appearance indicated a grave illness, and he was placed in bed in the Manhattan Eye, Ear and Throat Hospital, where myringotomy was performed under nitrous oxide anæsthesia. The incision was not followed by discharge of pus. Nevertheless he was irrigated with bichloride 1-10,000; and in the course of six hours a profuse discharge of pus appeared in the canal. The infective organism was the staphylococcus. For the two following days the temperature was 99° and the discharge very profuse. On the third day tenderness developed over the tip and antrum of the mastoid, with slight drooping of the superior wall of the external auditory canal. Headache persisted and the patient seemed unduly apprehensive.

Under gas ether anæsthesia a simple mastoid operation was performed, January 18th. The mastoid process, which was extremely dense, with few cells, contained much pus. Softened bone, which was adherent to the knee of the lateral sinus, was removed. The tegmen of the antrum was softened and adherent to the dura. The wound was packed with iodoform gauze; and the patient was returned to bed in good condition.

The case progressed satisfactorily for two weeks, when there was an unexplained rise of temperature to 102.° There was no chill, no sweating. The wound was clean and healing nicely. That night he complained of marked pain in the muscles of the calf of the right leg. There was no redness, but there was tenderness. The next day pain developed in the muscles of the left calf; and there was considerable pain in the sciatic notch on this side, with backache. The tongue was deeply furred. There was marked indicanuria. The temperature was 99.° Free purgation and aspirin controlled the pains to such an extent that the patient went to the toilet and walked about the hospital. On February 4th (the 17th day after operation) the temperature rose to 101°, and there was an exacerbation of his pains. He complained again of headache and that it hurt his eyes to read. Dr. Frank VanFleet pronounced the eye grounds to be normal. The temperature then became irregular, ranging from normal to 102°. The pain left the legs and centered in the sciatic notch and in the lumbar region. There was no Kernig, no Babinsky. The patellar reflex was normal. There was no stiffness in the neck. Dr. A. B. Duel was called in consultation, and confirmed my findings, viz, a clean, healing wound, with nothing but the fever pointing to involvement of the central nervous system. Dr. Charles H. Richardson confirmed my diagnosis of migrating muscular rheumatism. Free purgation and large doses of aspirin again gave considerable relief. The patient insisted on visiting the toilet, and seemed quite able to do so. The temperature continued to be irregular, ranging from 99° to

102.° The pulse varied with the temperature from 72 to 84; respiration 18 to 20.

On February 12th (the 25th day after operation) the patient was comfortable, and much less apprehensive than at any other time during his illness. The temperature was 100°, and he presented every evidence of a beginning convalescence. In the evening of this day he went unaided to the toilet, which was adjoining his room; and told me that he felt better than he had felt for two weeks. At about midnight his temperature rose to 102°; pulse was 108, respirations 104, pupils irregular. He gradually sank into unconsciousness, until at 5 A. M. (on the 26th day after operation) he was comatose. I opened the original mastoid wound. It was nearly healed. The lateral sinus seemed healthy. The dura was exposed and incised. The wound through it filled immediately with a great hernia of the brain. Careful search with a brain knife failed to reveal pus in the brain. A lumbar puncture brought forth a large amount of creamy fluid under great pressure. The patient died in a few hours, without having regained consciousness.

Autopsy was not allowed, but extensive examination through the wound failed to reveal an abscess. Much creamy fluid was recovered from the ventricles.

During the course of the illness there were frequent blood counts and frequent blood cultures, all negative. Widal and Wasserman were negative.

Examination of the cerebrospinal fluid resulted in the following report:

"Appearance,—mucopurulent, with considerable admixture of blood.

"Copper reducing substance—absent.

"Lactic acid test,—strong reaction.

"Cytologic examination.—About 10 per cent bulk of pus cells on centrifuging. These cells were largely polymorphonuclear leucocytes, with a few lymphocytes and endothelial cells.

"Bacteria.—In the stained smear were many chains of streptococcus mucosus capsulatus, with an unusually heavy capsule, so heavy as to leave their identity in doubt until stained by Gram's method. These grew freely on blood serum.

"Stain for tubercle bacilli,—negative.

(Signed) "J. G. Callison, M. D.,
"Pathologist."

G. T., male, age 19, was admitted to my service in the Manhattan Eye, Ear and Throat Hospital, December 5, 1912, with the following history: When an infant he fell, striking the back of the head. He was a delicate child, and did not develop well physically or mentally. The lack of mental development continued and he became an idiot. During early childhood he had measles, with a discharging ear which supposedly healed. Scarlet fever developed a few years later, accompanied by a discharging right ear. The discharge continued until the time of admission. No more complete history could be obtained because of the extreme ignorance of the patient's relatives.

Examination of the right ear revealed a large mass of spongy granulations nearly filling the canal; mastoid tender; deafness; no vertigo; no nystagmus. The temperature was normal.

A Schwartze-Stacke mastoid operation was performed under gas ether anaesthesia by Dr. S. McCullagh. The external auditory canal, middle ear and attic were found to be filled with granulations, and the ossicles were absent. The position of the dura was unusually low; and the lateral sinus was far forward. The antrum was absent. There was a large exposure of the dura and of the lateral sinus. The wound was packed and the patient was returned to bed. The next day the temperature rose to 102°, the patient became restless and complained of pain in the ear and in the back of the neck. There was no suggestion of central nervous involvement. The general condition was good. The temperature dropped to normal on the third day, and

for three days following pursued an irregular course, ranging from 99° to 101°. On the fifth day the temperature rose to 103°. The wound looked healthy and clean. The patient remained restless and persisted in rising from his bed. No rigidities were apparent. Babinsky and Kernig symptoms were absent. The eye grounds were normal. The blood culture was negative. Blood count: Leucocytes 18100. Large mononuclear leucocytes 5%; small mononuclear leucocytes 9%; polynuclear neutrophiles 86%.

For the three weeks following the temperature ranged irregularly from 100° to 102°. Other symptoms though sought for were not found. The eye grounds remained normal. The restlessness continued and the boy persisted in leaving the bed and in walking about. Typhoid fever and malaria were eliminated by blood examinations. Many of my colleagues in the hospital saw the case in consultation. Dr. Hutton, the attending physician, made this note on the 20th of December: "Deep-seated suppuration; if intracranial, so located as to give no definite localizing signs. Heart, lungs and abdomen negative." On December 28th, the boy was found wandering in the hall of the Hospital. On the following day there developed a fleeting external strabismus, and on the operated side slight facial paralysis. Ankle clonus and exaggerated knee-jerk demonstrable and Kernig's sign positive. Neck slightly resistent. The eye grounds were normal. A lumbar puncture revealed the cerebrospinal fluid, which was under pressure, to be almost creamy in color and consistency.

The evidences of central nervous involvement did not increase. A few hours before he died, he arose from his bed and appropriated articles belonging to other patients. He sank into coma an hour or two before death.

Autopsy revealed an unusually well-developed cerebrospinal lepto-meningitis.

Examination of the cerebrospinal fluid resulted in the following report.

"About 15 cc. recovered. 10 cc. sent to laboratory.

"Appearance,—greenish, creamy, muco-purulent.

"Amount of sediment,—2½ cc. in 10.

"Copper reducing substance,—absent.

"Lactic acid test,—very marked reaction.

"Cytological examination,—cellular elements are mostly polymorphonuclear leucocytes, with some lymphacytes and endothelial cells.

"Bacteria,—large numbers of either streptococci or pneumococci, with some bacilli resembling B. pyocyaneus. These cocci were culturally proved to be streptococcus mucosus.

"Stain for tubercle bacilli negative.

(Signed) "J. G. Callison, M. D.,
"Pathologist."

The post-mortem examination, conducted by Dr. Kopetzky, revealed the spinal cord covered with a thick, creamy exudate, which extended into the ventricles of the brain. There was no involvement of the lateral sinus. The dura was normal over the seat of the mastoid operation. The internal auditory meatus was filled with granulation tissue and pus.

I have reported these two cases not because cerebrospinal lepto-meningitis of otitic origin is a rare disease, but because in my experience, covering twenty-five years of otologic practice, I have not seen a single other case which did not present long before death some symptom other than fever pointing to involvement of the central nervous system. I confess myself at a complete loss to explain the lack of symptoms here presented. Brieger, quoted by Preysing, in the Transactions of the German Otological Association, 1912, says:

"In many cases of suppurative meningitis, we see all the symptoms disappear after opening the labyrinth and after repeated lumbar puncture. I have seen such cases, which appeared almost in the last stages, seemingly completely recover. They got up out of bed, walked about, felt perfectly well; and we were astounded to see that within a few hours after a condition of apparent complete well-being death should supervene. Autopsy revealed that in some cases there occurred in parts of the sub-arachnoid space inac-

cessible to lumbar puncture or to other exploratory methods, a wide-spread plastic infiltration; while in other cases there existed, in addition to these localized infiltrations, diffuse inflammation of the meninges."

ABSCESS OF FRONTAL LOBE OF THE BRAIN OF OTITIC ORIGIN, WITH EXHIBITION OF SPECIMENS.

By T. PASSMORE BERENS, M. D., New York, N. Y.

If the case of abscess in the frontal lobe of the brain that I am about to present to you is one of otitic origin, then,—so far as I have been able to learn from a comprehensive search of the literature reaching back to 1869, made by Dr. A. Braun,—it would seem to be unique. He has gathered together 49 cases, excluding the one that I present, of abscess in the frontal lobe, none of which was of otitic origin. Case No. 28, while presenting symptoms of mastoiditis, may be classified as a case of direct infection by extension from the ethmoidal labyrinth. I have given a short extract of each case, with its reference; but in order to save the time of this Society, I have also prepared a short table of the cases.

(1) *Frontal Sinusitis.* Early headache; apathy; rigidity of neck. Autopsy: Abscess frontal lobe.

(2) *Frontal and Ethmoidal Sinusitis.* Persistent headache; optic neuritis; pulse 55-60; somnolence. Recovered after operation; frontal lobe abscess.

(3) *Frontal Sinusitis.* Recovered after operation—frontal lobe abscess.

(4) *Frontal Sinusitis.* Early headache; pain; temperature 100½; increasing vomiting; incontinence urine; constipation. Autopsy: Abscess frontal lobe.

(5) *Pulmonary Tuberculosis.* Conjugate deviation; pupils dilated; temperature 100 1-5; delirium; stupor; hemiplegia. Autopsy: Multiple abscesses in frontal lobe.

(6) *Fractured Base.* Pain in eye; apathy; hemiplegia; vomiting. Autopsy: Abscess frontal lobe.

(7) *Unknown Etiology.* Headache; malaise; chills; temperature 99½. Autopsy: Abscess frontal lobe.

(8) *Fractured Skull.* Spasms of face extending to arm and leg; temperature 103. Autopsy. Abscess frontal lobe.

(9) *Healed Gun-shot Wound of Head.* Headache; pupils unequal; stupor; twitching and slight hemiplegia; dysphagia. Autopsy: Abscess frontal lobe.

(10) *Frontal Sinusitis.* Headache and vomiting; temperature 37½ c.; venous hyperaemia of eyes. Operation revealed abscesses frontal lobe. No autopsy.

(11) *Ethmoiditis.* Apathy; exophthalmos; delirium. Autopsy: Abscess frontal lobe. Basilar meningitis.

(12) *Ethmoiditis. Headache; vertigo; vomiting; apathy; hemiplegia.* Autopsy: Abscesses frontal lobe. Hemisphere adherent to lamina cribrosa.

(13) *Nasal.* Abscess frontal lobe.

(14) *Fracture frontal bone.* Apathy, incontinence of urine; prolapse of brain tissue, removed by thermo-cautery, opening anterior horn of lateral ventricle; 3½ months later bulging of scar; erotic mania and convulsions; spontaneous rupture. Disappearance of symptoms.

(15) *Frontal Sinusitis.* Abscess frontal lobe.

(16) *Frontal Sinusitis.* Multiple abscesses frontal lobe.

(17) *Panophthalmitis.* Temperature 39 C.; coma. Autopsy: Diffuse meningitis. Abscess frontal lobe.

(18) *Frontal Sinusitis.* Optic neuritis; coma; hemiplegia; headache. recovery after three operations for recurring abscesses in frontal lobe.

(19) *Trauma.* Headache; apathy; emaciation; hemiparesis; neuritis; pulse and temperature normal; symptoms of meningitis. Operation abscess frontal lobe. Recovery. One year later recurrence; unconsciousness; death. No autopsy.

(20) *Trauma of Orbit.* Convergent squint; apathy; loss of memory; later proptosis; choked disc; temperature subnormal; pulse 80. Operation, abscess frontal lobe. Recovery. Later, operation motor area, abscess. Recovery.

(21) *Tubercular Bronchiectatic Cavities.* Facial paralysis; Jacksonian epilepsy beginning right arm; hemiplegia. Autopsy: Two abscesses frontal lobe.

(22) *Tubercular Bronchiectatic Cavities.* Mental dullness; apathy; temperature 97-99; pulse 84; no other symptoms of brain involvement. Autopsy: Abscess frontal lobe.

(23) *Frontal Sinusitis.* Irritability; optic neuritis. Operation. Abscess frontal lobe. Recovery.

(24) *Frontal Sinusitis.* Slow cerebration; pulse 48. Operation. Abscess frontal lobe. Recovery.

(25) *Pansinusitis.* Operation. Epidural abscess. Frontal lobe abscess. Death.

(26) *Ethmoiditis.* Paralysis arm and leg; coma. Operation. Frontal lobe abscess. Death.

(27) *Frontal Sinusitis.* Intermittent headaches; convulsions. Two operations. Frontal abscess. Recovery.

(28) *Ozoena. Acute Mastoid. No Operation.* Symptoms: Mental dullness; headache. Autopsy: Abscess frontal lobe with dura adherent to ethmoid.

(29) *Frontal Sinusitis.* Headache; apathy; temperature subnormal; pulse 56. Operation. Autopsy: frontal lobe abscess.

(30) *Frontal Sinusitis.* Dullness of intellect. Operation frontal lobe abscess. Unconsciousness; hemiplegia. Second operation, motor area. Abscess found. Death.

(31) *Traumatic Ethmoiditis.* No localizing symptoms. Autopsy: Necrosis cribiform plate. Frontal lobe softened and infiltrated with pus.

(32) *Pansinusitis.* Headache; delirium. Operation, cyst of frontal lobe. Autopsy: basillar meningitis. Abscess, frontal lobe.

(33) *Frontal Sinusitis.* Stupor; vomiting. Operation. Frontal lobe abscess. Recovery.

(34) *Gun-shot Wound.* Headache; irritability; optic neuritis, left; retro-bulbar neuritis, right; increasing hemiplegia. Operation. Frontal lobe abscess. Recovery.

(35) *Traumatism.* Temperature 99 to 104; arm and leg paralyzed; retinal vessels tortuous; hemiplegia; coma; convulsions. Operation. Frontal lobe abscess.

(36) *Frontal Sinusitis.* Headache; one-sided convulsions. Operation. Abscess frontal lobe. Death.

(37) *Perforating Trauma.* Vomiting; general convulsions. Autopsy: Abscess frontal lobe.

(38) *Trauma.* Baby 10½ months. Severe pain; twitching and paralysis, arm and leg; fever; coma; pupils equally dilated; *circumference of head increased.* Autopsy: Abscess frontal lobe.

(39) *Trauma.* Vomiting; coma; tremor, one side. Autopsy: Abscess frontal lobe.

(40) *Trauma.* Twitching, arm and leg; epileptiform seizures. Operation. Abscess frontal lobe. Recovery.

(41) *Penetrating Trauma.* Temperature 39 C.; pulse 45; right hemiplegia; left ptosis. Operation. Abscess frontal lobe. Recovery.

(42) *Necrosis Frontal Bone.* Vomiting; fever; convulsions; anaesthesia of left face; diminished hearing, left; paralysis left sixth nerve; loss of speech; ulceration of cornea; coma. Autopsy: Extensive necrosis of frontal bone, purulent meningitis. Abscess frontal lobe.

(43) *Frontal Sinusitis.* Headache; convulsions; hemiparesis. Operation; brain hernia formed, then removed, liberating pus. Temporary relief, (one month). Convulsions 6 months; coma. Death. Autopsy: frontal lobe abscess. Purulent basillar meningitis.

(44) *Trauma of Orbit.* Convulsions. Operation frontal lobe abscess. Recovery.

(45) *Cause Unknown.* No paralysis; no convulsions. Clinical picture of tubercular meningitis. Frontal lobe abscess.

(46) *Trauma.* Headache; somnolence; vomiting; pulse slow; respirations slow; temperature normal; patellar reflexes diminished; pupils unequal; paresis left internal rectus; double optic neuritis; convulsions; pleurosthotonus. Operation. Abscess frontal lobe. Recovery.

(47) *Trauma.* Headache; somnolence; temperature 100; pulse 60; vomiting; Jacksonian epilepsy; hemiplegia; temporary aphasia. Autopsy: Abscess frontal lobe.

(48) *Acute Ethmoiditis.* Mentality dull; knee-jerk absent; optic neuritis. Autopsy: Pus in ethmoid. Abscess frontal lobe.

(49) *Orbital Abscess.* Headache; vomiting; pulse 64; temperature normal; Jacksonian epilepsy. Autopsy: Frontal lobe abscess and orbital abscess.

REDTENBACHER—"Hirnabszesse und Eiterung im Sinus frontalis nach Influenza." Wiener Medizinische Blaetter, 1892, 13.

Case 1. Female, 34 years of age. Had an attack of influenza. A few days later redness and swelling of left upper

eyelid. Then clonic spasms. Headache. Three weeks later, weakness of right half of body. Abscess of eyelid was opened. Did not heal. Apathy and rigidity of neck. Death three months later.

Post-mortem showed pus in the frontal sinus, with necrosis of its posterior wall. Dura adherent to this wall. At the apex of the frontal lobe, a nut-sized abscess.

A. DENKER—"Rhinogener Frontallappenabszess und extradualer Abscess in der Stirngegend, durch Operation geheilt." Archiv. f. Laryng. u. Rhin., 1900.

Case 2. Male, 17 years of age. Pain in region of left temple and eye. Foul odor from nose. Diagnosis of left frontal sinusitis and ethmoiditis was made. Intranasal operation. A month later, external operation. Two weeks later the headaches became worse, there was somnolence, there was blurring of the left optic disc, and the pulse was 55 to 60. The posterior wall of the frontal sinus was removed, and an epidural abscess was exposed. The dura was incised. Brain tissue was aspirated, and pus found. Brain incised and abscess evacuated. Recovery.

J. HERZFELD—"Rhinogener Stirnlappenabszess, durch Operation gehelit." Berliner Klinische Wochenschr. 1901, No. 47.

Case 3. Male, 20 years of age. Six weeks before admission he had coryza. Two weeks ago, left-sided frontal headaches. Diagnosis of left frontal sinusitis was made. External operation. Posterior wall of frontal sinus was found carious and removed. An epidural abscess was found. The dura was incised. A subdural abscess was evacuated. When this pus was wiped away, a fistula was seen in the frontal lobe of the brain. The fistula was enlarged with forceps, and a finger introduced into the abscess cavity. Iodoform gauze packing. Recovery.

E. S. STEESE—Medical and Surgical Report of the Presbyterial Hospital, 1898.

Case 4. I—C. S., 17 years of age. Six weeks ago sudden severe pain in both eyes, dimness of vision and vomiting. Pain lasted three weeks, and then vanished. Mental dullness.

delirium and stupor. Increasing difficulty in swallowing and breathing. Patellar reflexes absent. Pupils dilated. Pulse about 50, temperature 100.5° F. Vomiting, incontinence of urine, constipation, death.

Post-mortem showed frontal sinus disease. Abscess in left frontal lobe, ¾ inch in diameter. There was a perforation in posterior wall of frontal sinus.

Case 5. 2—C. S., 39 years of age. Phthisis for several months. Two weeks ago paralytic stroke, during sleep. Delirium and Stupor. Left hemiplegia. Occasional clonic spasm of left side, with spasmodic conjugate deviation. Right patellar reflex increased, and ankle clonus. Pupils dilated. Respiration stertorous. Temperature averaged 100.2° F. Death four weeks later.

Post-mortem showed an abscess one inch in diameter at upper end of fissure of Rolando. Another in occipital lobe. A large cavity in left frontal lobe.

Case 6. 3—A. H. Fracture at base of skull. Trephined in frontal region, and considerable fluid withdrawn. Six months later pain over right eye. Apathy and left hemiplegia. Patellar reflexes lost. Respirations shallow. Vomiting. Death.

Post-mortem disclosed a large abscess in right frontal lobe. A few foci of suppuration were also found at the top of the left parietal lobe.

Case 7. 4—B. J., 25 years of age. One week ago headache, malaise and chills. Temperature averaged 99.5° F. Death after a week.

Post-mortem showed flattening of the convolutions of the brain, and an abscess one inch in diameter in left frontal lobe. Entire lobe softened. Etiology unknown.

Case 8. 5—Female. Was struck by cable car and received a linear fracture of the skull. Spasms commencing in left side of face and extending to arm and leg. No paralysis. Temperature averaged 103° F. Duration three days. Death.

Post-mortem disclosed two large abscesses in the frontal lobe. Laceration of cerebellum. Pneumonia. Muco-pus in smaller bronchi of both lungs.

Case 9. 6—M. S., 45 years of age. Bullet removed from head ten months ago. Headache, stupor, twitching of right hand and leg, and slight paralysis of left side. Dufficulty in swallowing. Right pupil dilated, and left contracted. Rigor. Death.

Post-mortem.—Entire right frontal lobe honeycombed with abscess cavities.

M. PAUNZ—"Ueber den rhinogenen Hirnabscess." Arch. f. Lar. u. Rhin., 1902-03, vol. 13.

Case 10. Female, 16 years of age. Acute left frontal sinusitis. Headache and vomiting. Temperature 37.5° C. Pulse 60 to 66. Dilated sluggish pupils. Venous hyperaemia of the eyegrounds. Operation. Small frontal sinus was found. Dura was exposed and found bulging. Aspirated pus through dura. Incision of dura and brain-substance. About 80 cc. of pus evacuated. Death six months later.

H. MAAS—"Zur Casuistik und Therapie der Gehirnabszesse nach eigenen Erfahrungen." Berliner Klin. Wochenschr. 1869.

Case 11. Male, 45 years. Nasal polypi. Removed several times. Frequent nose-blood. Apathy. Vertigo. Naso-pharyngeal polyp. This, as well as the nasal polypi, removed. Patient was somewhat relieved. Followed after several days by increase in the apathy. Exophthalmos, delirium and death.

Post-mortem showed dura at the base of the skull covered with pus. The lamina cribrosa and upper surface of the sphenoid were destroyed. Abscess in the frontal lobe of the brain, above ethmoidal region.

D. JACUBASCH—"Abszess im vorderen rechten Gehirnlappen." Berliner Klin. Wochenschrift, 1875.

Case 12. Male, 20 years. Had sore in nose. Dull headache, vertigo and vomiting. Speech slow and difficult. Pulse 56. Evening temperature 39° C. For several weeks no change. Then apathy. Pain in back of neck. Left-sided hemiparesis. A month later death.

Post-mortem—Right hemisphere adherent to lamina cribrossa. Large abscess in right frontal lobe. A second abscess further back.

Case 13. SEIFERT—"Abscess of frontal lobe of nasal origin." Revue hebd, de laryngol, etc. 1905.

BORCHARD—Arch. f. Klin. Chirurgie, 1904, vol. LXXIV.

Case 14. Female, 8 years. Was kicked in left side of forehead by horse's hoof. Frontal bone fractured, Brain tissue exposed. Loose fragments of bone removed. Dressing. Child is apathetic. Passes urine involuntarily. Prolapse of brain-tissue removed with thermo-cautery. This opens up anterior horn of lateral ventricle. Patient discharged six months later. Three and a half months later she was readmitted. Five days before her readmission the old scar began to bulge. She developed symptoms of erotic mania, and had several convulsions. The swelling ruptured spontaneously and evacuated pus. This was followed by disappearance of the mental symptoms. The swelling with recurrence of the mental symptoms reappeared several times in the course of the next few years.

Case 15. CISNEROS—"Abscess of the frontal lobe following frontal sinusitis." Revue hebd. de laryngol, etc. 1905.

Case 16. R. HOFFMAN— "Multiple abscesses of the brain, resulting from bilateral traumatic frontal sinusitis." Arch. internat. de laryngol. 1906.

W. REIS—"A case of panophthalmitis with brain abscess and fatal meningitis." Arch. f. Augenheilk., 1905.

Case 17. Male, 37 years of age. Was struck in left eye with stick a week before. Panophthalmitis. Eye-ball was incised and pus evacuated. Five days later, severe headache, temperature 39° C, coma, and death the same evening.

Post-mortem showed a diffuse meningitis, and an abscess in the anterior part of the frontal lobe.

L. B. RAWLING—"Case of Chronic Abscess of the Frontal Lobe." Transactions of the Medical Society of London, 1907.

Case 18. Male. Frontal headache with oedema of the right upper eyelid, in May, 1905. Roof of orbit was exposed, and found bare of periosteum. On June 1, coma, and paralysis of left side of body and face, vomiting, opetic neuritis, slow pulse and subnormal temperature. Skull was opened so as to expose outer and anterior aspect of frontal lobe, and an abscess was found. Rapid recovery. Discharged cured in the middle of July. In 1906, return of the same symptoms. A similar operation was performed, and another abscess found. Recovery. On October 13, 1906, he was admitted for the third time. Marked bulging at the site of former operations. Drowsy and irritable. Severe headache. Left hemiplegia. Abscess was not found. Marked hernia. On October 24, again operated, and abscess found. Recovery. At a later date frontal sinus was opened and found to contain pus and carious bone.

F. L. TAYLOR—"A Case of Abscess of the Frontal Lobe of Traumatic Origin." N. Y. Medical Journal, May, 1908.

Case 19. Male, 27 years of age. Was struck with a brick above the right eye in May, 1906. Was not rendered unconscious. A month later, superficial abscess was opened. Later, headache, apathy, emaciation, optic neuritis, slight paresis of the left side of the face, and weakness of grip in left hand. Stiffness of back of neck, vomiting, incontinence of urine and feces. Pulse and temperature normal. Operation showed abscess in frontal lobe on right side. Recovery, with poor vision. In October of the following year he became uncon scious, had several convulsions, and died.

COULTER—Ophthalmoscope, 1909.

Case 20. Female, 24 years of age. Piece of wood, 1½ inches long, was driven into the left orbit, through the upper eyelid. Removed. This was followed by a convergent squint and diplopia. Swelling in upper outer angle of orbit. Incised. Three months later, apathy, loss of memory, pulse 80, temperature subnormal. Both eyes were proptosed. Left external rectus was paralyzed. Choked disc. The skull was trephined behind the external angle of the left orbit. Abscess was found in frontal lobe. A month later, right arm and

leg began to twitch. Trephined over motor area, and another abscess tapped. Cure.

SCHORSTEIN—Lancet, 1909.

Case 21. 1—Male, 33 years. Tuberculosis, with bronchiectatic cavities. Coma. Facial paralysis left. Muscular spasm, beginning in right arm. Seven days later, hemiplegia of right side. Three days later, death. Two abscess were found in frontal lobe, on autopsy.

Case 22. 2—Male, 21 years. Tuberculosis, with bronchiectatic cavity. Dullness and apathy. No other symptoms of brain involvement. Pulse 84. Temperature 97° to 99° F.

Post-mortem showed abscess in right frontal lobe.

L. V. CARGILL—WM. TURNER—ST. CLAIR THOMSON—"A Case of Abscess in left Frontal Lobe Originating from Suppuration in left Frontal Sinus." Proceedings of the Royal Society of Medicine, 1907-1908.

Case 23. Male, 31 years. Patient had an orbital cellulitis, which was incised. Fourteen days later, irritability and optic neuritis. Frontal sinus was opened and posterior wall found necrotic. Brain covered with granulations. Abscess in frontal lobe evacuated. Recovery.

M. P. LAURENS—Proceedings of Parisian Society of Lar., Otol. & Rhin., abstracted in Journal of Laryngology, 1908.

Case 24. Slow cerebration, pulse 48. Chronic frontal sinusitis. Operation. Posterior bony wall intact. Infection of brain, either through venous or lymphatic channels. Abscess in frontal lobe, size of mandarin. Recovery.

L. M. HURD—Transactions of Amer. Lar., Rhin. & Otol. Society, 1910. Discussion to Dr. McCoy's paper.

Case 25. Chronic pan-sinusitis, epidural abscess, frontal lobe abscess,, meningitis and death.

J. McCOY—Transactions of Amer Lar., Rhin. & Otol. Society, 1910.

Case 26. 1—J. L., 1½ years of age. Ethmoiditis. External operation. Osteamylitis of vertical plate of frontal bone. Epidural abscess. About three weeks later, paralysis of

right arm and leg. Ten days later, coma. Frontal lobe opened. Large quantity of pus evacuated. Death.

Case 27. 2—H. E., 10 years of age. Frontal sinusitis left. External operation was done by an eye surgeon. Two months later necrotic bone in posterior wall of frontal sinus was removed. Dura covered with granulations. This was followed by intermittent headaches. Two months later, convulsions. Large abscess found in frontal lobe of brain. Five days later ,another connecting abscess was found. Counter opening was made in temporal region, and through and through drainage instituted. Recovery.

THOMAS HUBBARD—Trans. of Amer. Lar., Rhin. & Otol. Society. 1903.

Case 28. Woman, 25 years of age. Ozaena. Acute mastoiditis. No operation. Two months later, mental dullness and headache. Mastoid tenderness and death. Autopsy showed dura adherent to under surface of right frontal lobe. Small abscess in centre of frontal lobe.

FAGGE—Guy's Hospital Reports, 1909, vol. 63.

Case 29—J. R., 60 years of age. Empyema of frontal sinus. Radical operation. Headache, apathy, subnormal temperature and pulse of 56. Operation showed abscess in frontal lobe on left side. Death.

Post-mortem showed abscess extending from orbital surface of frontal lobe nearly to anterior horn of lateral ventricle.

J. A. WHITE—Transactions of Amer. Lar., Rhin. and Otol. Society. 1910. Discussion to Dr. McCoy's paper.

Case 30. Chronic frontal sinusitis. No symptoms of brain involvement, except some dullness of intellect. Operation evacuated frontal lobe abscess. Following operation, unconsciousness. Two days later, lost use of right arm and muscles of face. Opened another abscess in region of Rolandic fissure. Death.

STUCKY—Annals of Otol., Rhin. and Lar. June, 1907.

Case 31. Traumatic ethmoiditis, with meningitis and abscess of frontal lobe. No localizing symptoms.

Post-morten showed no disease of pia or dura, except over cribiform plate, which was necrotic. Fracture through inner table of left frontal sinus. Anterior and middle cerebral convolutions softened and infiltrated with pale green pus.

STUCKY—Cleveland Medical Journal, March, 1911.

Case 32. Female, 49 years of age Pansinusitis. Posterior wall of frontal sinus necrotic. Dura exposed and covered with granulations. Headache and delirium. Improved and was well for three months after operation. Then recurrence of pain. Dura opened and cyst of frontal lobe found containing 1½ ounces of thin straw-colored fluid. Died of sepsis four months later.

Post-mortem. Dura thickened and adherent to brain, in frontal and tempero-sphenoidal region. Base of brain bathed in cloudy fluid. Entire right frontal lobe contained large cavity filled with creamy fluid. Walls of cavity ¼ inch thick. Anterior half of right tempero-sphenoidal lobe also disintegrated.

HANS RISCHE—Zeitschr. f. Ohrenheilk., vol. 62.

Case 33. Male, 19 years. Headache and swelling above left eye. Incised and pus evacuated. Temperature 37° to 38° C. Pulse 60. Stupor, vomiting. No pus in nose. External frontal sinus operation. Posterior wall of frontal sinus showed small perforation. Dura covered with granulations. Dura incised. Abscess in frontal lobe evacuated. ½ to 2 drachma. Recovery.

FOSTER KENNEDY—Journal of Nervous and Mental Diseases, 1911.

Case 34. Struck over right fronto-parietal region with spent bullet. Bullet entered bone, but did not penetrate cranial cavity. Six months later, headache and irritability. Optic neuritis on left side. Slight left facial weakness. Cerebrospinal fluid normal. Wasserman reaction in fluid positive. No history of syphilis. Loss of smell on right side. Retrobulbar neuritis of right side. Gradually increasing left hemiplegia. Operation showed large abscess in frontal lobe. Recovery.

ERNEST SACHS—Journal of Nervous and Mental Diseases, 1911.

Case 35. Boy, 4 years. Two weeks before admission, fell, and struck on forehead. Dull and irritable. On day of admission right arm and leg became suddenly paralyzed. Temperature 99° to 104° F. Lumbar puncture gave clear sterile fluid. Three days later, unconsciousness, and convulsions more marked on the right side. Vessels of left retina tortuous. Operation. Frontal lobe abscess.

W. FREUDENTHAL—International Journal of Surgery, September, 1908.

Case 36. Miss J. S., 25 years. Acute right frontal sinusitis. Headache, and nasal discharge. Oedema of right eye. Killian operation. Posterior wall of sinus eroded. Dura bulging. Dura incised. Two ounces of pus evacuated. Following operation several attacks of convulsions on left side. Death several days later. No autopsy.

YOUL—Lancet, 1847, abstracted by Holt, Arch. of Pediat., 1898.

Case 37. Child, 8 months. Received wound in temporal region with some sharp body, which healed. Two weeks later, vomiting and general convulsions. Died in one hour. Autopsy showed abscess containing 2 ounces of pus, in frontal lobe, at point opposite scar of external wound. Surrounding brain tissue much disorganized. Abscess communicated with lateral ventricle.

WYSS—Jahrb. f. Kinderheilk. IV. Abstracted by Holt in Arch. of Pediatr.

Case 38. Boy, 10½ months. Fell several feet and struck on head. Four days passed before first symptoms occurred. Sudden onset with crying and severe pain at night, followed in 10 hours by twitching of right arm and leg. Eight days later, paralysis of right arm and leg. Fever. Circumference of head increased. Six weeks after onset, coma. Pupils equally dilated. Six to eight convulsions daily, during one of which the child died.

Autopsy showed left hemisphere larger than right, owing to an enormous abscess involving almost the entire frontal

lobe. No communication with ventricles which were distended with clear serum.

KOEHLER—Irrenfreund, Heilbrunn, 1879, Vol. XXI. Abstracted by Holt in Arch. of Pediatrics 1898.

Case 39. Boy, 18 months. Fell, striking head against a file, which penetrated frontal bone. Four weeks later scar was inflamed, and piece of file, 1 inch long, was removed. Later, vomiting and coma. Small swelling appeared at aperture in frontal bone, which pulsated, and was easily reduced. Pus removed by aspiration and patient improved. Coma again developed, but upon aspiration no pus. Tremor of left half of body. Death in coma.

Post-mortem. Abscess size of egg in left frontal lobe, communicating with opening in frontal bone and with left lateral ventricle. Pus had penetrated through the foramen of Munro into right ventricle. Third and fourth ventricles also contained pus.

BORELIUS—Jahrb. f. Kinderheilk., Vol. XXXII. Abstracted by Holt in Arch. of Pediatr., 1898.

Case 40. Boy, 4 years. Fell upon splinter of wood, which punctured upper eyelid. One week later lid swollen and epileptiform seizures and other cerebral symptoms. No paralysis, but twitching was more marked in left arm and leg. Deep incision was made in upper eyelid, and large amount of pus evacuated. Probe passed through roof of orbit into frontal lobe. Drainage tube inserted and patient recovered. Discharged in one month.

ZDEKAUER—Jahrb. f. Kinderheilk., 1893. Vol. XXXV. Abstracted by Holt in Arch. of Pediatr., 1898.

Case 41. Boy, 3¾ years. Fell upon nail, which penetrated left side of head. Five days later fever, right hemiplegia and left-sided ptosis. Temperature 39 C. pulse 45. Over left temporal bone, wound from which pus discharged. Operation performed, during which dura was found to be fluctuating. Puncture of dura released 2 ounces of green foul-smelling pus. Drainage tube inserted. Recovery, except for paresis of left hand.

REINER—Jahrb. f. Kinderheilk., Vol. XI. Abstracted by Holt in Arch. of Pediatr., 1898.

Case 42. Male, 3 years. Rickets. Cerebral symptoms of six weeks duration. Bone disease for two and a half months. Then vomiting, fever and convulsions. Headache of left side and anæsthesia of left side of face. Diminished hearing on left side and paralysis of left sixth nerve. Convulsions and high fever, loss of speech and loss of consciousness. Cornea ulcerated and perforated. Death in 10 days.

Post-mortem showed large abscesses occupying nearly whole of left frontal lobe. Turbid serum in ventricles. Purulent meningitis at convexity, and on left side of base. Extensive necrosis of frontal bone. Dura was adherent here and greatly thickened.

TREITEL—"Ueber Hirnabszesse nach Stirnhœhleneiterung." Deutsche Med. Wochenschr., 1898, Vol. XXII.

Case 43. Female, 22 years of age. In March, 1894, nasal obstruction, purulent discharge from left side of nose and pain in forehead and cheek. Polypi were removed from left side of nose on several occasions, as well as middle turbinate. In December, 1894, severe headache on left side of forehead. External operation on frontal sinus. Posterior wall was rough. Pain did not disappear after the operation. In April, 1895, she was operated by Dr. Rotter. He found dura exposed over an area of one cm. A larger exposure was made and the brain punctured. No pus was found. On May 20, repeated convulsions. Hemiparesis of right side. Pulse normal. Pupils equal and reacted well. Eyegrounds normal. On May 22, repeated punctures of brain were made. No pus found. Brain hernia followed. After two weeks, portions of this became gangrenous. This was removed and base cauterized. About a drachm of pus escaped. On August 6, patient was discharged, although wound still secreted pus and the hemiparesis remained. On September 11, she was readmitted and frontal sinus again operated on. In October, convulsions. In March, 1896, the ethmoids were removed. On March 21, death during a convulsion.

Post-mortem. Over left frontal lobe dura adherent to brain. Large abscess in frontal lobe. Basal surface of frontal lobe hemorrhagic. Purulent basillar meningitis.

J. M. LEE—"Abscess of Frontal Lobe of Brain and Convulsions Due to Accidental Puncture of the Orbital Roof." Homeopath. Eye, Ear and Throat Journal, 1897, Vol. III.

Case 44. Male, 12 years. Fell on stub of weed, which penetrated right upper eyelid, and passed through sphenoidal fissure into frontal lobe of brain. Wound healed without medical treatment. Six months later, convulsions. Small suppurating point on lid incised, and finally a piece of the stub worked out. Wound healed. A year later spasms reappeared, and lasted three weeks. Cicatrix again opened, and piece of stub removed. He was not troubled for nearly a year, when convulsions returned, and lasted six months. A third incision revealed no foreign body. Spasms subsided again. A year later they reappeared. Eyegrounds normal. A U-shaped flap was made on forehead. Two buttons of bone were removed by trephine. Dura bulged and showed a small opening through which sero-pus discharged. Dura was incised, and cavity was seen in frontal lobe. Drainage. Spasms disappeared in one week.

HANOT—Archiv. Gen. de Med., Paris, 1890.

Case 45. Large abscess of frontal lobe, without paralysis or convulsions, giving during life the clinical picture of tubercular meningitis.

WM. MacEWEN—"The Pyogenic Infectious Diseases of the Brain and Spinal Cord."

Case 46. 1—Boy, 11 years of age. Was admitted on May 9, 1889. Ten weeks before he was struck by a stone on the forehead, an inch above the left eyebrow. The boy was unconscious for half an hour. The wound healed in three weeks. Beginning four days after the injury, there occurred headaches, which gradually increased in intensity. For six weeks past, somnolence and repeated attacks of vomiting. About a month before admission there occurred, after the vomiting, profuse perspiration. Pulse was slow and weak; respiration slow and irregular; temperature

normal. Patellar reflexes were diminished. Pupils were unequal, the left the smaller. Reaction normal. Slight paresis of the left internal rectus. Bilateral optic neuritis. On May 17, convulsions in both arms, but most marked in the right. Then pleurosthotonus to left side. Operation without narcosis. Incision through the scar disclosed carious bone. This was removed and an area of thickened dura was exposed. Dura was incised, showing pia adherent to it, and inflamed. An aspirating needle encountered pus in the frontal lobe an inch from the surface. Abscess opened, and decalcified bone drainage tube inserted. Immediately after operation there was an increase in rapidity of pulse and respiration. Consciousness returned. Gradual improvement of all symptoms.

Case 47. 2—Boy, 11 years of age. Admitted in 1876. Two weeks before admission he fell, and struck his forehead upon the floor. Six days later, headache. On admission wound, 1½ inches long, was seen above left eyebrow. Roughened bone could be felt at the bottom. Somnolence. Rectal temperature, 100° F., pulse 60 to 65. Six days later, vomiting. The following day, chills. In the evening, temperature 103° F. Five days later, i. e., 26 days after the injury, convulsions, beginning in the right side of the face and extending to the right arm and leg. This followed by paralysis of the right side, and aphasia. The aphasia lasted an hour, then disappeared. Paralysis improved after several hours. Diagnosis was made of abscess in third frontal convolution, between speech centre and internal capsule. Operation was refused. Two days later, death.

A partial post-mortem was allowed. A plate of bone was removed from the temporal region, the upper margin of which corresponded to the third frontal convolution. An extradural exudate was disclosed, and hyperæmia of the dura. The dura was incised and reflected, and the brain incised. At a depth of 1 inch from the surface an abscess was encountered.

R. T. WILLIAMSON—"A Case of Abscess in the Right Frontal Lobe of the Brain." Medical Chronicle, 1890-91.

Case 48. Male, 19 years. Admitted March 22, 189 . On February 18, influenza with profuse nasal discharge, and frontal and occipital headache. Mentality dull. This dullness became progressively more marked. Knee-jerks absent. Optic neuritis in right eye. Temperature between normal and 106.4° F. ante-mortem. Death on April 4.

Post-mortem. Large abscess in anterior part of right frontal lobe. A small amount of pus in right ethmoidal cells.

J. C. RENTON—"Notes on a Case of Cerebral Abscess, Subsequent to Orbital Periostitis." Glasgow Medical Journal, 1886.

Case 49. Male, 12 years. Admitted October 17, 1884, for what appeared to be a right orbital cellulitis of 10 days' duration. On October 22, an incision was made along the inner side of the upper eyelid, with evacuation of pus. On October 28, severe headache, pulse 64, temperature normal. Vomiting. On November 11, spasms of left side, which soon became general and ended in death on the same day. Temperature was never higher than 99° F., being generally 97.6° F.

Post-mortem. Convolutions over right frontal lobe, slightly flattened. Right orbital plate necrosed. Dura inflamed over this area. Abscess in anterior half of right frontal lobe. Abscess cavity lined by distinct membrane. There was no direct communication between the pus from the orbit and the cerebral abscess.

Case 50. J. S., male, age 27, entered my service at the Manhattan Eye, Ear and Throat Hospital on November 10, 1912, suffering from slight deafness in the right ear of seven years duration; intermittent purulent discharge; pain in the right ear for two weeks; mastoid tender; drooping superior wall of canal. Mother died of apoplexy at the age of 52. The patient never had had any serious illness. Venereal disease denied. Occupation, glass blower and prize fighter.

On November 12th, under gas ether anæsthesia, I performed a Schwartze-Stacke radical operation. The mastoid

was partly pneumatic, partly eburnated, and contained much pus. The middle ear and the attic contained much granulation tissue, and the malleus and the incus were necrosed. The stapes was located and was not disturbed. Neither the dura nor the sinus was exposed. A flap was made and the wound sutured behind. Packed with sterile gauze.

The case pursued an uneventful course, and was transferred at the end of eight days to the out-patient department for dressings.

On November 28th, 16 days after the operation, while being dressed in the clinic by a member of the House Staff, the patient had a sudden gush of blood from the middle ear cavity. The cavity was quickly packed with iodoform gauze, but the bleeding was not controlled. The hemorrhage was profuse, flowing through the gauze packing in a stream off the chin. It was checked by Dr. Stucky, the House Surgeon, who repacked firmly with iodoform gauze. The patient was placed in bed. He was weak and pale, but the pulse was of fair quality. Temperature remained below 100°. A full dressing was done on the fourth day after the hemorrhage, when the wound looked clean.

On the morning of the fifth day (December 4th) at 5.30 A. M., the patient had a convulsion, lasting from five to six minutes. He was not seen by a doctor during this convulsion. The nurse described it as "severe," "the patient shaking all over, the head retracted, with frothing at the mouth and gripping at the bed clothes with both hands." The House Surgeon arrived as the convulsion was over. He described the condition as follows: "Cold perspiration over the entire body. Both pupils markedly contracted, equal and reacted to light. The patient could not be aroused to answer questions. The pulse was full, strong, regular, 84; temperature, rectal, 102°. Respirations 34. A full dressing was done and the wound looked healthy."

Fifteen minutes later there was another convulsion, lasting for a few minutes. This was also over before the House Surgeon could be summoned. At 9 A. M., the tem-

perature was 99°, respirations 22, pulse 112. At 3 P. M., the temperature was 100.8°, respirations 24, pulse 116. Blood count: Leucocytes, 15,000; polymorphonuclear leucocytes, 89%; small lymphocytes, 7%; large lymphocytes, 4%. Wasserman positive on December 10th. Blood culture negative. Spinal fluid under high pressure, but clear and "non-meningitic," according to chemical tests by Drs. Kopetzky and Callison. Color clear, copper reducing substance present. Lactic acid, marked reaction. Noguchi test weakly positive. Hydrochloric acid precipitation test negative. No polymorphonuclear leucocytes in stained smears. Culture negative. Urine negative, except for faint trace of albumen. Eyes,—fundi, normal. (Drs. Webster, Thomson and Van Fleet). After the convulsions, the patient lay with eyes closed and drawn expression, but usually quiet. He complained only of headache, and if asked how he felt, he replied, "all right, except for a dull headache." Asked to localize the headache, he said that it was "over the whole right side of the head and forehead." The headache persisted, and the patient very gradually became stuporous. The whole right side of the head became tender to percussion. He did not answer questions promptly, but up to two hours before he became comatose, answered intelligently. He remembered his name, age, address, and could give a good history of his trouble. The temperature never went higher than 102°, nor lower than 99⅘°. The pulse was never lower than 80, until December 10th, when it varied between 60 and 78—always full and regular. On December 6th, Kernig was negative, Babinsky negative, no ankle clonus, no stiff neck. On December 10th, Kernig positive, Babinsky positive, the neck stiff, no ankle clonus. On December 11th, Kernig positive, Babinsky positive, ankle clonus very marked and equal, the neck stiff. The patient had an attack of typical Jacksonian epilepsy, beginning in the toes of the left foot and extending over the whole left side.

The patient was at once placed on the table, and the middle cerebral fossa was exposed by removing the tegmen. Incision through the dura gave 2-3 ounces thick, creamy,

foul pus, which seemed to flow from the anterior limit of the incision. The brain tissue protruded rapidly until a hernia

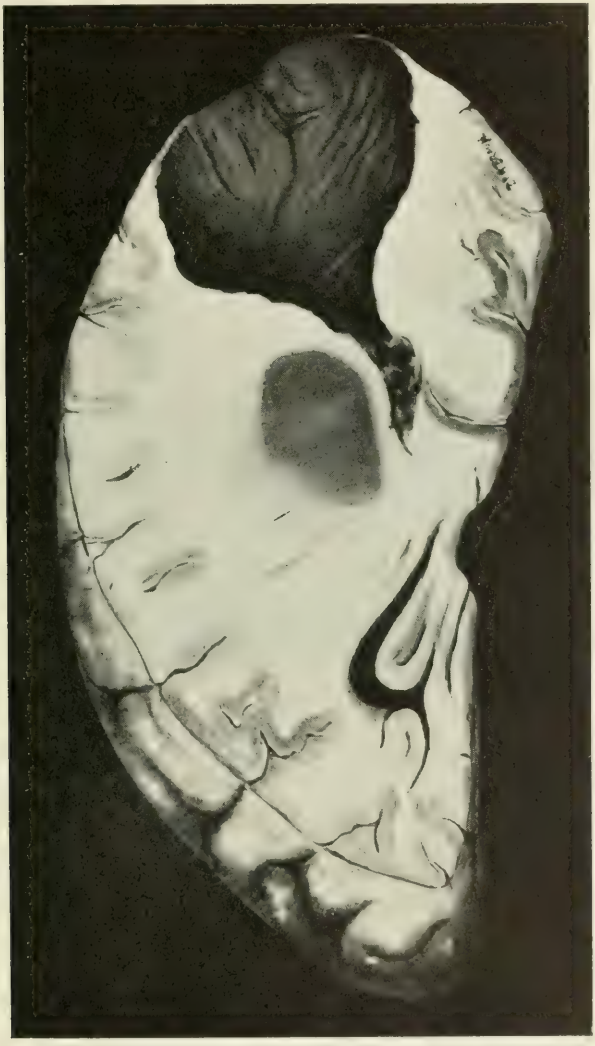


Fig. 1.

of the brain filled the bone wound. Examination of the brain substance with a knife in all directions, failed to locate

any deep abscess. A cigarette drain was inserted. After the operation, temperature was 102°, pulse 124, respirations 22, intravenous saline infusion on the table. The patient never regained consciousness. The temperature remained between 102 and 103 until December 15th, just before his death.

Autopsy by Drs. Braun and Friesner and Stucky. There was a large subdural abscess over the right hemisphere extending posteriorly to the posterior limit of the parietal lobe. There was a large abscess, (see Fig. 1), the size of a hen's egg, thick-walled, full of greenish-yellow foul pus, occupying

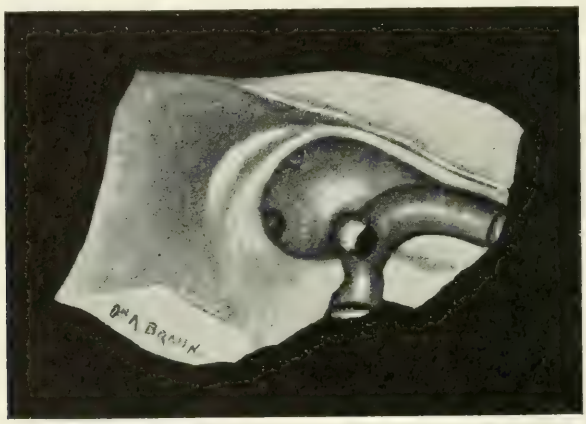


Fig. 2.

most of the right frontal lobe; this abscess had burst and drained into the subdural space, and probably was the source of the subdural abscess. The accessory sinuses of the nose were absolutely normal. The internal carotid artery contained a clot and was eroded at the knee, where there was a dehiscence of the bone. (See Fig. 2).

This case presents several points of great interest to me.

First. There was no doubt, early in the case, that there was intracranial pressure; and early exploration of the brain was not undertaken because there was no localizing symptom. The attack of Jacksonian epilepsy was the first localizing symptom.

Second. Exposure of the dura through perfectly healthy bone of the tegmen of the antrum and the attic revealed what at the time of operation was supposed to be a superficial abscess in the tempero-sphenoidal lobe of the brain.

Third. The presence of a large, deep abscess was strongly suspected because of the rapid formation of the large hernia of the brain, and the brain was thoroughly, though fruitlessly explored.

Fourth. The finding of an unsuspected abscess in the frontal lobe; the fact that it had burst, and the fact that the pus from it was the cause of the subdural abscess.

Fifth. The pus from the abscess showed no growth after 48 hours on Loeffler's blood serum.

Sixth. The presence of a dehiscence in the bony canal of the internal carotid artery and the presence of a perforation through the walls of the carotid artery at the site of the dehiscence in the bone.

Seventh. The occurrence of a severe hemorrhage from the internal carotid 14 days after operation.

This case may not have been of otitic origin, but I believe it to have been. It seems reasonable to suppose that the dehiscence in the bone of the carotid canal allowed of an erosion and infection of the walls of the artery, and that the abscess was the direct result.

One of the most constant localizing symptoms of frontal lobe abscess is hemiplegia. In the case under discussion, this symptom was undoubtedly due to the extension of the subdural abscess over to the motor area. In some of the other cases reported, the hemiplegia was due either to the abscess involving the internal capsule, to the accompanying encaphalitis which involved the internal capsule, or to pressure of the abscess upon the motor area.

Discussion.

Dr. Emil Amberg, of Detroit, Mich., thought it significant that the streptococcus mucosus was found in both of the cases reported. This organism acted, as Dr. Cutler char-

acteristically expressed it, like a thief in the night. The Vienna school has emphasized the danger of the same. It has been claimed that the patient who suffers from a streptococcus mucosus infection of the ear might get along comfortably, e. g., after a mastoid operation, and suddenly he might develop meningitis.

Dr. Ewing W. Day, of Pittsburg, Pa., had found, in chronic cases, a slight indefinite malaise. In one or two in which he performed the radical mastoid operation lumbar puncture was made when the patient was put on the table, and, without any signs of meningitis, the cerebrospinal fluid was found loaded with the microorganisms of the disease. The patient had had only a little earache before. In chronic purulent otitic disease meningitis might be present for a long time without being suspected. Since finding these cases he had done lumbar puncture before the radical mastoid operation in every instance.

Dr. Berens, in closing the discussion, referred to a paper by Dr. Dickson, the pathologist of the New York Eye and Ear Infirmary on X-ray diagnosis in these cases. The report covered a number of years, and stated that in all fatal cases the *streptococcus mucosus* was present.

THE PROTECTIVE MASTOID OPERATION: AN OPERATION OF ELECTION.

By W. SOHIER BRYANT, A. M., M. D., New York, N. Y.

The point of view concerning the function of the mastoid operation has undergone a striking change. Formerly the entire idea of this operation was stern necessity—the last resort for the saving of life. In its present state of perfected technic, the mastoid operation usually accomplishes this function. New technical skill and common sense have perceived in it a wider scope, a range of usefulness adapted to the supreme help of the ear.

The improvement in accuracy of diagnosis, certainty of prognosis and results of technic, justifies an extension of the field of usefulness of the mastoid operation. Formerly the mastoid operation has been usually an emergency operation; the protective operation is an operation performed at the time of election. The protective mastoid operation is a supplement to nature in her work of economy and conservation.

The protective mastoid operation should not be performed without profound technical deliberation. It is an axiom that we do not operate in every case of middle ear suppuration for many cases of purulent middle ear inflammation convalesce satisfactorily without an operation. This is especially true in chronic middle ear suppuration where many cases will heal with little difficulty and with very satisfactory results by appropriately applied milder treatment. As a general rule the cases that indicate a protective operation are those whose pathological condition is so far advanced that it seems unavailing to attempt milder treatment and also in the cases which do not respond to mild treatment within a reasonable time.

The protective mastoid operation is definitely indicated in the following.

(1) All cases which have some residual hearing in the presence of middle ear suppuration resistant to mild treatment. The hearing will become progressively worse as long as the destructive process continues because of the extension of ulceration and increased middle ear cicatrization.

(2) Cases of middle ear suppuration (with or without mastoid complications), which are the source of chronic toxic absorption. The protective operation, by checking the suppuration of the ear, destroys this nidus of infection.

(3) Cases of middle ear suppuration (with or without mastoid complications), which may be the focus of infection causing serious complications, such as brain abscess, sinus thrombosis or meningitis.

An appreciable interval between the inception of the middle ear suppuration and the performance of the protective mastoid operation should be allowed in order to justify our conclusion that, in the case under consideration, the suppuration will not stop without a protective operation or in time to save the hearing and also that the danger of serious complication is imminent. Proper diagnostic skill and due weighing of accurately ascertained diagnostic indications negate the chance of error in the election of this protective operation.

The ideal protection of the ear cells for the mastoid operation is as follows:

I. The mastoid operation for the cure of chronic middle ear suppuration.

II. The mastoid operation for the cure of acute or subacute middle ear suppuration with or without mastoid involvement.

III. The mastoid operation to relieve the patient from a long and dangerous expectant period while waiting for resolution or for development into a mastoid abscess sufficiently urgent to require an emergency operation.

The special technic to be employed varies with the stage of the suppuration, its extent and the topography of the region involved.

First Group: In cases of chronic middle ear suppuration, the various forms of radical mastoid operations are protective operations because:

They destroy the focus of infection of the discharging ear which supplies pyogenic organisms disseminated by means of linen, dust, hand shaking and in various other ways. A source of infection is thus destroyed which might have become a source of infection and which might have given rise to every grade of pyogenic infection, to the most virulent.

They destroy the source of bacterial poisons, which are often the source of chronic toxic absorption, as is seen in cases with chronic middle ear suppuration. The protective mastoid operation interrupts this morbid cycle giving the individual a chance to recover normal health after the suppuration of the ear has been corrected by the protective operation.

They arrest chronic middle ear suppuration and prevent terminal complications, such as brain abscess, sinus thrombosis, meningitis, broncho-pneumonia, bacteremia, nephritis, pericarditis, endocarditis and erysipelas.

Various technics have been devised for the mastoid operation for the radical cure of middle ear suppuration. They are all founded on a common principle, namely the obliteration of the mastoid antrum. Different technics have been advocated by Schwartze, Stacke, Heath, Bondy, Mahu, Streit, Stacke, Luc, Barany, Citelli and others.

I prefer the technics that destroy the least tissue and of them I prefer the technic that I have adopted in either the conservative radical operation or the modified radical operation.

The conservative radical mastoid operation is indicated when the middle ear structures are of no further auditory value or are destroyed. In my conservative radical mastoid operation the middle ear is not curetted and no tissue is removed from the middle ear. The antrum is opened widely into the auditory canal. The outer anterior wall of the attic is removed with its contents. The Eustachian tube is pre-

ferably kept open. The restoration of a cicatricial drum membrane is not hindered in any way.

The results in this conservative radical mastoid operation are: An arrest of suppuration, a stable middle ear cicatricial condition, no painful dressings, a shortened convalescence, no disfigurement and taking into consideration the loss of the middle ear mechanism, a maximum of hearing. In this operation, the hearing is improved beyond what it was before the operation.

The modified radical mastoid operation is adapted to the radical cure of chronic middle ear suppuration when the middle ear sound transmitting mechanism is still capable of some functional activity.

The operative field is attacked as in the conservative radical but the attic is not obliterated; it is opened only as far as the preservation of the ossicles in position will allow.

The results in my modified operation are, in many respects, similar to those obtained in the conservative radical mastoid operation. In this modified operation, however, it is possible to obtain a shorter convalescence; and, since the election of this operation presupposes some residual functional activity of the middle ear mechanism, it is possible to obtain a higher degree of hearing, often above normal. In this operation also, the hearing is improved beyond what it was before the operation.

Second Group: The mastoid operation is indicated as a protection in many cases of acute or subacute middle ear suppuration with or without mastoid complications, when the indications point to eventuation into a chronic middle ear suppuration which, for its final cure, will require a radical mastoid operation.

Under these circumstances, the indications for the operation are based on the Roentgen-ray findings. All acute cases of middle ear suppuration should be sciogramed. Where the sciogram shows a mastoid bone without air cells surrounding the antrum, we are reasonably certain that in these cases the suppuration of the middle ear will not readily yield to treatment. Furthermore we are practically sure that the

acute suppuration will eventuate into chronic suppuration if indeed it is not brought to an abrupt termination by the development of the fatal complication of mastoiditis. After the inception of middle ear suppuration in dense mastoids an interval of from four to eight days should intervene before doing my modified radical mastoid operation. Cheatle's anatomical observations form the basis of the indications.

The technic of the operation for the cure of acute suppuration of the middle ear with the solid mastoid bone is the same as the technic of my modified radical operation.

The results of my modified radical operation in acute cases of middle ear suppuration in solid mastoid bones are: Arrest of suppuration, stable middle ear condition, no painful dressings and a moderately short convalescence. Also the results are better for the hearing, and in proportion to the previous group, more cases are restored to normal or above. In many cases the hearing is improved even beyond what it was before the suppuration of the middle ear.

Third Group: In acute or subacute infections of the middle ear, the protective mastoid operation is indicated to relieve the patient from a dangerous expectant period while waiting for a mastoid abscess to develop or the suppuration to resolve. This group is limited to the cases in which the sciogram reveals pneumatic cells communicating with the antrum. The most important indication for the operation is unsatisfactory middle ear drainage, even after myringotomy and douching.

A protective mastoid operation is advisable in order to cut short the process because of the danger to the patient from capital complication of middle ear infection which may arise at any time without warning. The protective mastoid operation is also indicated on account of the impairment to hearing caused by the middle ear suppuration. The longer the continuation of the ulcerative process, inevitably associated with this suppuration, the greater the loss of the essential parts of the sound transmitting mechanism of the middle ear. The longer a suppuration lasts and the greater

the destruction of tissue, the more abundant will be the final cicatrices which hamper the physiological sound transmission of the middle ear.

After the inception of middle ear suppuration in cellular mastoids, an interval of from 7 to 14 days should intervene before doing my simple mastoid operation.

The technic of my simple mastoid operation demands the removal of the mastoid process, the opening of all cells, the removal of all affected bone, the removal of the available posterior osseous meatal wall between the anulus tympanicus and the facial ridge, the leveling of the edges of the bone wound and the longitudinal section of the membranous canal along the posterior inferior wall, the closure of the posterior wound with the insertion of a minimum drain followed by its early removal, that is to say, my modified blood clot dressing.

The results in my simple mastoid operation are the same as those in the previous group, except that the minimum time of convalescence is reduced to as low as three days, and that the hearing is more frequently restored to normal or above, and in still more cases is improved beyond what it was before the suppuration of the middle ear.

The object of these three elective operations—conservative radical, modified radical or simple mastoid—is:

From a pathological standpoint,—to stop the suppuration.

From a protective standpoint,—to forestall complication.

From a functional standpoint,—to preserve or improve the hearing.

The supreme test of the technic in a specialty of any definite organ is the conservation of the function of that organ. Our specialty is too often criticised as accomplishing too little in the preservation of the hearing. The mastoid operation offers, when used according to the indications, the most effective method for the seasonable amelioration of the consequences of persistent suppuration. Thus, in addition to the important function of stopping the suppuration, the elected operation, by providing the maximum protection to the parts of the hearing mechanism, preserves the hearing which is in jeopardy.

AUTHOR'S BIBLIOGRAPHY.

Technic and Results

Some Modifications in the Operative and After-treatment of Mastoiditis. N. Y. Eye and Ear Infirmary Annual Report, Jan. 1906. Vol. XII. pp. 81-91.

Exhibition of a Patient Operated on for Mastoiditis, Complicated by Epidural Abscess. Transactions of the Amer. Otolog. Soc., 1906. Vol. X., Pt. 2., pp. 354-355.

Operative Technique and After-treatment for Mastoiditis with Epidural Complications. Med. Record, Mar. 31, 1906. Vol. 69., No. 13., pp. 502-505.

A Case of Mastoiditis. Archives of Otol., April, 1906. Vol. XXXV., No. 2., p. 131.

The Radical Mastoid Operation Modified to Allow the Preservation of Normal Hearing. Transactions of The Amer. Otolog. Soc., 1906. Vol. X., Pt. II., pp. 292-295. Also N. Y. Med. Jrl., Oct. 20, 1906. Vol. LXXXIV., No. 16 (Whole No. 1455), pp. 780-781.

Modified Blood Clot in Mastoid Surgery. Annals of Otol., Rhinol. and Laryngol., Sept. 1906. Vol. 15, No. 3., pp. 489-492. Also Archives Internat. de Laryngol., d'Otol. et de Rhinol. (Paris), Sept.-Oct. 1906. Tome XXII, No. 2., pp. 501-504.

Radical Mastoid Operation. Archives Internat. de Laryngol., d'Otol. et de Rhinol., Sept.-Oct. 1906. Vol. XXII., No. 2., p. 592.

The Conservation of Hearing in Operations on the Mastoid Region. Annals of Otol., Rhinol. and Laryngol., March, 1907. Vol. XVI., No. 1., pp. 32-5. Also Boston Med. and Surg. Jrl., March 7, 1907. Vol. 156., No. 10., p. 300.

The Use of the Blood Clot in Radical Mastoid Cases. Archives Internat. de Laryngol., d'Otol. et de Rhinol., March and April, 1907. Tome XXIII., No. 2., p. 586.

Rapid Convalescence after Mastoid Operations. The Laryngoscope., April, 1907. Vol. 17, No. 4, pp. 273-277.

The Technic of the Complete Mastoid Operation Improved. Shortened and Simplified Through the Digastric Route. Transactions Amer. Otolog. Soc., 1907. Vol. X., Pt. III., pp. 461-463. Also Annals of Otol., Rhinol. and Laryngol., Dec. 1907. Vol. XVI., No. 4., p. 871.

A Case of Mastoiditis and Epidural Abscess: Operation and Rapid Recovery. Transactions Amer. Otolog. Soc.,1907. Vol. X., Pt. III., pp. 503-509. Also Annals of Otol., Rhinol. and Laryngol., Dec. 1907. Vol. XVI., No. 4., p. 873.

The Radical Mastoid Operation. Indications for and Results from its Employment. N. Y. Med. Jrl., June 29, 1907. Vol. LXXXV., No. 26., (Whole No. 1491)., p. 1213.

Mastoideotympanotomy: The Operation of Election for Persistent Otorrhea and Acute Mastoideo-tympanic Ostitis. Post-Graduate Quadri-Centennial. Vol. 1908., p. 228.

Report of all the Mastoid Operations Performed by the Author in the Service of Dr. Robert Lewis, Jr., at the N. Y. Eye and Ear Infirmary in 1907. N. Y. Eye and Ear Infirmary Report, July 1908. Vol. XIII., pp. 24-43.

A Case of Streptococcus Encapsulatus Aural Infection and Modified Radical Mastoid Operation. Archives of Otol. Feb. 1908. Vol. XXXVII., No. 1., pp. 69-72 and p. 75.

Presentation of Three Cases Showing Functional and Cosmetic Results after various Mastoid Operations. Annals of Otol., Rhinol. and Laryngol., 1910. Vol. XIX., p. 221.

Technical Points which furnish the best Curative, Functional, and Cosmetic Results in Mastoid Operations for Extradural Lesions. Amer. Jrl. of Surgery., Feb. 1910. Vol. 24., No. 2., pp. 47-51.

The Modified Radical Mastoid Operation. Transactions Amer. Laryngol., Rhinol. and Otol. Soc. Meeting Atlantic City, N. J., June 2, 1911.

The Modified Radical Mastoid Operation for the Cure of Otitis Media Purulenta Chronica. Med. Record, New York, Nov. 16, 1912, Vol. LXXXII., pp. 886-888, and XI International Otological Congress Transactions, 1912.

Opportunities for the Preservation of Hearing. Jrl. of the A. M. A., Mar. 22, 1913. Vol. LX., No. 12., p. 878.

Clinical Indications for Mastoid Operation. Annals of Otol., Rhinol. and Laryngol., 1913.

BIBLIOGRAPHY OF RADICAL MASTOID OPERATION AND ITS MODIFICATIONS.

Barany. Meine Methode der conservativen Radikaloperation bei chronischer Mittelohr-Eiterung. Medizin. Klinik. 1912. No. 18., p. 726.

Bondy. Totalaufmeisselung mit Erhaltung von Trommelfell und Gehörknöchelchen. Monatsschrift f. Ohrenheilkunde, 1910. Vol. 44, H. 2, pp. 15-23.

Operation radicale conservatrice. 81 Cong. des Naturalistes et Médecins Allemande. Abst. Archives Internat. de Laryngol. d'Otol. et de Rhinol., Jan.-Feb. 1910. Vol. 29., pp. 230-233.

Bryant. A New Instrument for Mastoid Surgery. (Case I). Transactions American Academy of Ophthalmology and Oto-Laryngology, 10th Annual Meeting, Buffalo, Sept. 14-16, 1905. Also Laryngoscope, Oct. 1905. Vol. 15, No. 10., pp. 796-801.

Some Modifications in the Operation and After-treatment of

Mastoiditis (Case I). The N. Y. Eye and Ear Infirmary Annual Report. Jan. 1906. Vol. 12., pp. 81-98.

Presentation of a Case of Modified Radical Operation. Archives of Otology, Feb. 1906. Vol. XXXV., No. 1., p. 38.

Presentation of a Case of Mastoiditis. Archives of Otology. April, 1906. Vol. XXXV., No. 2., p. 131.

Presentation of a Case of Modified Radical Operation. Archives of Otology, Aug., 1906. Vol. XXXV., No. 4, pp. 398-400.

Abstract of Radical Mastoid Operation. Archives Internat. de Laryngol., d'Otol. et de Rhinol, Sept.-Oct., 1906. Vol. XXII., No. 2, p. 592.

The Radical Mastoid Operation Modified to Allow the Preservation of Normal Hearing. Transactions Amer. Otolog. Soc. Meeting June 26-27, 1906; Vol. X, Pt. II., pp. 292-295. Also N. Y. Med. Jrl., Oct. 20, 1906. Vol. LXXXIV., No. 16, pp. 780-781.

The Modified Radical Mastoid Operation for the Cure of Otitis Media Purulenta Chronica. Med. Record, Nov. 16, 1912. Vol. LXXXII., pp. 886-888. Also XI Internat. Otological Congress Transactions.

Citelli. Ueber die konservative Radikaloperation. Archiv. Internat. de Laryngol., 1912. Tome 33, No. 2, p. 362. Also Monatschrift f. Ohrenheilkunde, 1913. Vol. XLVII., p. 63.

Heath. The Cure of Chronic Suppuration of the Middle Ear Without Removal of the Drum and Ossicles or loss of hearing. The Lancet, Aug. 11, 1906, p. 353.

Cases Illustrating the Successful Treatment of Chronic Suppuration of the Middle Ear Without Removal of the Drum or Ossicles or Loss of Hearing. The Lancet, Jan. 26, 1907, p. 221, and Apr. 27, 1907, p. 1146.

Kashiwabara. Operative Freilegung der Mittelohrräume vom Gehörgang aus Archiv f. Ohrenheilkunde, 1911-1912. Vol. 87, p. 20.

Luc. Evidement partiel avec conservation de la membrane du tympan. Bull. d'Ito-Rhin.-Laryngol, Paris, 1911. Vol. XIV., p. 167.

Mahu, G. De l'evidement pétro-mastoidien partiel avec conservation du tympan et des osselets, Ann. d. mal, de l'Oreille, 1910. Vol. XXVI., No. 9, pp. 245-258.

Totalaufmeisselung mit Erhaltung von Trommelfell und Gehörknöchelchen. Soc de Laryngol., d'Otol. de Paris, März, 1911.

Ein Fall von Totalaufmeisselung mit Erhaltung der Gehörknöchelchen bei einem Kinde. Internat. Ctrlblt. f. Ohrenheilkd., 1912. Vol. X., No. 4, p. 165.

Maraspini. Cinq cas d'évidement pétro-mastoïdien avec modification de la technique. Abst. A. I. de L. I. & R., 1910. Vol. XXX., No. 3, p. 954.

Schwartze. L'ouverture opératoire de l'apophyse mastoidienne. Congrès Périodique International des Sciences Médicales, 1886. Tome IV., p. 53.

Die operative Eröffnung des Warzenfortsatzes. Handbuch der Ohrenheilkunde, 1893. Vol. II., p. 815.

Schwartze and Eysell. Ueber die künstliche Eröffnung des Warzenfortsatzes. Archiv f. Ohrenheilkunde, 1873. Vol. VII., p. 157.

Stacke. Indicationen, betreffend die Excision von Hammer und Amboss. Internat. Med. Kongress, Berlin, 1890. Vol. 33, p. 120. Also Archiv f. Ohrenheilkunde, 1891. Vol. 31, p. 201.

Weitere Mitteilungen über die operative Freilegung der Mittelohrräume nach Ablösung der Ohrmuschel. Berliner Klin. Wchschrft, 1892. Vol. 29, No. 4, p. 68. Also Archiv f. Ohrenheilkunde, 1892. Vol. 33, p. 120.

Über Konservative Radikaloperationen. 20th Meeting of German Otol. Soc., Francfort, June, 1911. Internat. Ctrblt. f. Ohrenheilk, 1910-1911. Vol. IX., p. 448. Also Verhdlg. d. dtsch. Otol. Gesellschaft, 1911. Vol. XX., p. 343.

Conservative Radical Operation on the Middle Ear. Annals of Otol., Rhinol. and Laryngol., Dec. 1911. Vol. XX., No. 4, p. 893.

Streit. Ueber die operative Freilegung von Antrum und Kuppelraum mit Erhaltung des Trommelfells und der Gehörknöchelchenkette. Monatsschrift f. Ohrenheilkd., Berl., 1910. Vol. 44, pp. 1229-1241.

PRESERVATION OF THE ANTRAL CAPSULE IN OPERATIONS FOR UNCOMPLICATED ACUTE SUPPURATIVE PROCESSES OF THE MASTOID.

By NORVAL H. PIERCE, M. D., Chicago, Ill.

What is meant here by the antral capsule or box? It is that portion of the temporal bone bounded above by the diploetic bone and pneumatic cells lying between the antral cavity and the inner table of the skull; anteriorly by the interposed pneumo-diploetic bone and the compact substance of the external auditory canal; the outer wall by the same character of bone, through which run the vessels between the antrum and cortex; posteriorly by the compact substance sent in from the roots of the zygomatic process, small (minute) pneumatic spaces, and the walls of the sigmoid sinus; and inferiorly by the cribriform plate, if we may so call it.

While this capsule is not by any means as clearly differentiated from the rest of the bone as is the capsule of the labyrinth, yet it possesses a resistance to inflammatory and softening processes, to which the mastoid is subject.

If we look at the floor of the antrum, we find innumerable minute openings which communicate with the pneumatic spaces in the mastoid. These minute pneumatic outlets may be divided into an anterior and a posterior group by a buttress of hard bone, which is sent in to the substance of the mastoid process from the inferior root of the zygomatic process. I have never seen a description of the buttress in literature. It is much more marked in compact mastoid processes, but even in the pneumatic type it may be demonstrated. The buttress ends some distance from the top of the mastoid, being replaced here by diploe or pneumatic spaces.

In the large majority of acute inflammations in the mastoid the softening process which eventuates in abscess formation, spreads from the antral floor downwards along the minute openings of the anterior group of cells, first form-

ing a fistula inferiorly, or below the antrum, which is bounded anteriorly by the hard substance of the external auditory canal and posteriorly by the buttress of compact bone before-mentioned. The fistula later passes beneath the buttress of bone posteriorly to the knee of the sigmoid sinus.

In all acute inflammations, it is necessary in operative procedures to remove all osseous tissue which is incapable of reorganization and, in a broad way, this may be determined by the scoop.

In a series of cases operated on in the last eighteen months, a method of operation has been pursued which has certain distinct advantages. We were led to this mode of operating by rather unfortunate phenomena which occurred in the after treatment of certain cases of acute mastoid inflammation, in which the anterior, posterior and inferior walls of the antral box were removed; namely, recurrent inflammations within the greatly depressed mastoid scar, and prolonged after-treatment, owing to a persistent fistula caused by spreading downward of the muco-periosteum from the aditus which necessitated several curettements and eventually, a much depressed deforming scar. Especially in children is this apt to occur.

It has been noted on the operating table that the antral box or capsule, with the exception of the cribriform floor, presented a very marked resistance to the softening process found elsewhere in the mastoid bone. In other words, the perforation of the mastoid antrum, if approached in a certain way to be described later, was found to be almost invariably through the floor and downward. If we destroy the box which contains the antrum and curette out the softened tissue there, it has been noted that the mucosa from the middle ear spreads much more rapidly over the osseous structures than the delimiting granulation tissue springs from the inner tablet of the skull. That is to say, if we remove the upper wall of the mastoid box to the inner tablet of the skull, the anterior wall to the compact bone of the external auditory canal, the posterior wall to the sigmoid sinus and all of the floor, leaving only the hard cancellous structure, poorly sup-

plied with blood vessels, then this mucosa spreads to an enormous extent and we have a permanent, large, dependent cavity lined with muco-periosteum of very low resistance and vitality; indeed, it is to the spreading of this mucosa down into the cavity into which we pack in after-treatment, which interferes with the closure of some of these cases; it is only when we have removed this by a curettement and were fortunate enough to have the surrounding granulation tissue spring up to close it over, that we bring about eventual healing.

The mode of operative procedure is as follows: The external incision and reflection of the soft parts are carried out as in all mastoid operations. Instead, however, of first making entry through the mastoid fossa or the suprameatal triangle and entering the antrum from the side, we proceeded from below, taking off the cortex of the bone about half an inch from the tip upward to the lower margin of the mastoid fossa, forward to the hard bony substance of the external auditory canal, and posteriorly about a finger's breadth from this. The soft bone is scraped out and the buttress of hard bone, which was mentioned before as being thrown into the mastoid profess from the lower wing of the zygoma, is removed, and a search is made for the fistula, which is almost invariably found descending from the cribriform plate of the mastoid antrum downward between the hard, bony substance of the external auditory meatus and the hard buttress of bone before mentioned. Just sufficient of the softened bone is removed from the bottom of the antrum to admit our drainage tube, and then we proceed to remove whatever other diseased portion of mastoid presents itself, posteriorly to the posterior cells upwards to the squamus cells or zygomatic cells but always being careful to preserve the antral box and not opening into it other than at the extreme inferior portion, as before-mentioned. Of course, in simple mastoid operations, we cannot explore the cells of the floor of the tympanic cavity or of the roof or of the tubal cells. It is only in radical operations that we can gain access to these. But these are of an entirely different series of cells

from the mastoid cells, and do not empty into the mastoid antrum. The tube extends to about half an inch below the mastoid incision.

When we have cleared away the softened tissue, wherever it may have been found, and have explored the walls of the sinus, a piece of a small infant's catheter is inserted into the antrum through its floor, and the bone cavity in the mastoid is packed about the tube with xeroform gauze.

If the meninges or the sinus have not been exposed, we sew up the entire wound, with the exception of a small portion left open for the gauze and the tube at the inferior margin of the wound. If the meninges have been exposed, then only a few stitches are taken at the upper margin sufficient to cover the healthy denuded bone with periosteum.

The external auditory canal is packed with a strip of xeroform gauze, and a voluminous fifty per cent. alcohol dressing, covered with oiled silk, is then placed over the wound. The external dressing is changed daily. On the third or fourth day we irritate the antrum and the cavum through the catheter and on the fifth day the xeroform packing is removed.

If at this time it is found that the packing from the external auditory meatus is dry, the tube from the antrum is removed and only a thin strip of gauze is inserted up to the opening into the antrum.

The advantages of this simple mode of operating are:

1. Rapidity of healing, the after-treatment being lessened in the majority of cases by many days or weeks.

2. The avoidance of ugly, disfiguring scars.

3. The avoidance of a large permanent cavity or fistula formed by the undue spreading of mucosa from the middle ear into the mastoid, causing recurrent otitis media or recurrent mastoiditis in the former or prolonged after-treatment in the latter case.

It is unnecessary to add that this method of operating is not adapted to acute cases in which labyrinthine or intracranial complications are present. Here the antral box must be sacrificed in our exploration.

Discussion.

Dr. Edward B. Dench, of New York City, had observed the spreading of the mucous membrane of the tympanic cavity in exenteration in simple cases. This was the first time the subject had been brought before the society. This spreading of the mucous membrane from the tympanic cavity into the operative cavity explained delayed healing in many cases. He had found a firm limiting wall to the antral box in many cases, and deep infiltration toward the middle intracranial fossa. He did not understand how Dr. Pierce could eliminate these cells if he opens from below. The paper was of distinct value in that it called attention to a simple operation. He had often told his assistants that he had fewer complications when he did less radical surgery. There was evidently much yet to be learned regarding technic.

Dr. H. Burke Blackwell, of New York City, asked how many cases had been operated upon in this manner, and if the aural discharge persisted after operation.

Dr. Stephen H. Lutz, of Brooklyn, N. Y., asked if it is not a fact that the antrum is now more frequently attacked from below instead of by the old classic operation, in other words, that the operation is started from the tip cells instead of from the triangular space. While this was perhaps the first time attention had been called to this point, as a matter of fact many otologists had been doing the same thing. In attacking the bone from below one is apt to find the fistula that Dr. Pierce mentioned; the rest of the operation consists in removing the diseased bone.

Dr. Kopetzky:—The question put to me by Dr. Richards requires an answer not in the form of my personal opinion on the methods advocated by Dr. Pierce but rather a discussion of the principles of pathology involved. While I am upon my feet, it is also proper that I refer to another

aspect of this same subject which Dr. Blackwell recently presented before the Otological Section of the New York Academy of Medicine, and which I see he is also to present before this society today. The same principles are involved in both papers.

I hold with Panse, Boenninghaus and others including Schiebe, that in the acute bacterial inflammations of the middle ear and mastoid process that there is a very marked infiltrate and inflammatory swelling of the mucous membrane lining the tympanic and mastoid antral cavities. The space inside of these cavities which normally contains air, is filled with purulent detritus. This mucous membrane should not be removed. With the removal of the purulent accumulations in the mastoid process and the opening of the membrana tympani as is done in the precedure advocated by Dr. Pierce, this membrane is conserved.

When recovery takes place and this membrane has been removed then it is replaced necessarily by connective tissue. When recovery takes place and the membrane has *not* been removed, then there results a status which more nearly approached the normal conditions. This is better accomplished by the method which Dr. Pierce has shown today, than by the operative procedure which Dr. Blackwell advocates. I do not want to discuss fully the stand of Dr. Blackwell at this time, but I do want to indicate the directly opposite stands that these two men are taking.

Finally the study of the cases which recur among children, has been proven to be in a large measure due to the removal of the membrane lining of the mastoid antrum, and such recurrences are less likely when that membrane is left in situ. For all these reasons I may answer Dr. Richards by saying that although I do not favor absolutely following the technique given by Dr. Pierce, yet according to my knowledge of the pathology and some experience with cases that have recurred and required secondary operations, I believe that he is upon the right road, and the principles underlying the operation he advocates are sound.

Dr. B. Alexander Randall, of Philadelphia, thought the understanding of the matter under discussion could be considerably clarified by remembering the development of the parts concerned. The tubo-tympanic cavity opens out from the pharynx, and forms the three-fold tympanic space, of the attic, atrium and antrum, as demonstrated by Leidy. Every aural pneumatic space must be developed from these spaces already formed. There are the mastoid cells, the zygomatic cells, and others of the squama growing out from the attic, while tubal cells tend to grow past the carotid into the petrous bone. There are large cells sometimes along the internal meatus, although textbooks state that there are no cells in this region. The mucous membrane in the pharynx and tympanic cavity differs little from that in the cells. These are absolutely continuous structures, not only in their configuration but in their nature.

So respecting the point made by Dr. Pierce as to the buttress of bone. After the antrum is fully formed before birth it is a question whether it ever enlarges, except as its walls become a network of openings, so that it is difficult to say just what its size is. In the infant it is bounded externally by what Leidy has called the scute (German, schupe), and it is only in some instances that this remains distinct and separable throughout life. Yet often there is this layer of non-porous bone which represents the sutural line although it more commonly is permeated with cells, which it can develop just as other parts of the squama can develop cells. This squamous plate and the mastoid portion of the petrous bone coalesce, as a rule. There are beautiful specimens of non-union in the Wistar museum in Philadelphia. A limited "mastoid operation" can be done through this squama plate, without touching the mastoid at all. There is this line of demarcation which Dr. Pierce showed.

Dr. Randall believed that when the blood-clot operation was used more extensively there would be less to worry about in the mastoid operation. Healing would be just as quick and the operation would be modified only by the diseased bone to be dealt with.

Dr. Pierce, in closing the discussion, emphasized the point which he wished to make in the paper, viz., the fact that if one drain below these cells it is not necessary to remove the anterior wall. If a small perforation is made these dependent cells will drain themselves. This bone has enormous power of regeneration. It is not, as has been suggested, an incomplete operation, it is a very complete operation. There is no reason why one cannot go into the squama, the zygoma, or anywhere one pleases, if a certain amount of bone around the antrum is preserved. This buttress of bone mentioned extends into the zygoma. In eleven cases operated upon in this way he had seen no discharge from the middle ear.

FURTHER OBSERVATIONS ON THE TECHNIQUE IN THE AFTER-CARE OF THE RADICAL MASTOID OPERATION.

By PHILIP HAMMOND, M. D., Boston, Mass.

To achieve progress in any endeavor, it is necessary to keep constantly in mind the goal toward which we are striving. Under no condition should we allow our efforts to be diverted from the main object by attempts, however zealous, to prove the truth of pre-conceived notions, unless those notions lead in the most direct way to the solution of the questions under consideration.

Pedantic as this may seem, it strikes directly at the cause of many exploited "methods"; for often one who has been instrumental in the development of an idea, or of some operative procedure, will become so fixed in his habits that he will tolerate no change in his accustomed routine, even though inconceivably superior methods have been adopted by his co-workers.

The advisability, nay even the necessity of constantly changing one's mental attitude toward the treatment of the radically operated mastoid has been impressed upon the writer during an experience covering practically twenty years; if we but analyze the progress that has been made in this time, we shall see that it has come about by attempts to accomplish a definite result in the simplest possible way.

Even with the first cases, it was recognized that success could not be attained without epidermization of the cavity, and to this end, a post-auricular opening was left through which various cutaneous flaps were introduced to aid the epidermis from the meatus in spreading throughout the inside of the bone.

Following the tendency of that time, the wounds were packed almost daily with gauze; and, needless to say, the granulations thrived upon this treatment. Then, to aid in

the process of epidermization, small Thiersch grafts or islands of epidermis were introduced. The permanent opening behind the ear proved so disfiguring that it was soon abandoned.

Opinions still vary as to certain details of the complete radical which will be taken up later, but authorities are agreed up to a certain point. It is generally conceded that the bone operation should be most thoroughly and painstakingly performed, all crevices smoothed out, and all projections removed. Some form of plastic operation is then performed upon the posterior part of the cartilaginous meatus, to serve the double purpose of free access to the cavity for inspection and treatment and to furnish a dermal covering for a part, at least, of the mastoid. The type of this operation varies with the demands of the case and the habits of the operator but the tendency is to obtain a meatus that will be sufficiently large to readily allow of all after treatment of the entire cavity. The posterior wound is now generally sutured throughout with the object of obtaining healing by first intention. The method of suturing that has given by far the best results in the hands of the writer is by means of the small metal clamp or Michel suture. Not only is the apposition much better than that generally obtained, but they can be applied much more rapidly than ordinary sutures. Only one failure has been noted in an extended series, and that was in a case that had been subjected to repeated operations. With the object of preventing haemorrhage from the incision, a dilute solution of adrenalin chlorid was injected subcutaneously over the operative field as a preliminary, after the method of Jansen. There was a general breaking down of the wound and some sloughing; but this could not properly be attributed to the method of suture.

From this point on we encounter the greatest diversity of opinion as to the proper course to follow. Roughly speaking, the exponents of the various methods may be divided into two classes: those who use the skin graft to aid Nature, and those who do not.

Before attempting to take any stand on the subject, we should remember, first that our whole aim is to favor the complete epidermization of the cavity, and secondly that this should be accomplished as speedily and with as little distress to the patient as possible. The question then rapidly narrows down to one of results. Which method, or combination of methods, is most likely to show a favorable outcome? In this query we are likely to be led astray by prejudice, or by the fact that familiarity with a certain routine may have made the operator an expert with that particular method. Under these conditions, any observer can scarcely avoid a bias toward the procedure which produces the best results at his hands, although the skill with which he administers his technique may be more of a factor in the successful issue than the means adopted.

To counteract this tendency, and to overcome as far as possible the objections which necessarily obtain where the operative work is carried on at the hands of one man, some of my colleagues at the Massachusetts Charitable Eye and Ear Infirmary have joined with me in an attempt to search out the truth of this matter. Cases have been operated upon by all the methods and the results noted. In addition to this, as many as possible of the cases operated upon for several years past have been requested to report at the hospital for observation. These cases have been carefully examined, and the results freely discussed by advocates of the different methods of operation; so that the cases have been subjected to the criticism of the opponents as well as of the partisans of the methods employed.

As a result of this inquiry, several facts seem to have been established; first, the end results do not appear to favor any particular procedure, although the length of treatment was lessened in the cases subjected to grafting. Second, the cases in which the primary graft was used apparently did as well as those in which it was applied at a later stage. Third, as was to be expected, the best results were observed in cases where the most careful bone work had been done.

One of the interesting observations arising from a study of this series was the comparison of the very thorough exenteration practiced today with that done several years ago; the most recent cases allow of much better access to the middle ear, and the complete removal of the facial ridge leaves an extremely smooth cavity. This naturally aided in the healing of the wound, particularly where skin grafts were used. Many of the older cases had prominent ridges left at the site of the posterior canal wall, not only greatly interfering with the inspection of the ear but actually handicapping the recovery.

Another point on which improvement has recently been made is in adopting some device to keep the meatus permanently enlarged. In a majority of the older cases it had narrowed down so that it did not provide room for thorough inspection of the cavity. In the hands of the writer, the best results have been obtained by the use of a Koerner flap. This is carried well into the concha, the base of the flap being made as wide as possible. The resulting tongue is then pulled forward out of the meatus, and the skin of the flap separated from the cartilage, much as a submucous operation is done in the nose. The cartilage is now removed from the flap to its base. A deep buried chromicized gut suture is then passed through the base of the flap from behind, and caught into the periosteum of the posterior edge of the wound, thus holding the meatus well open. To overcome the rather pointed corners in this artificial meatus, a short piece of large calibre rubber tubing is slit lengthwise, so that it is in cross-section like the letter C and can be expanded to fit any size canal. This is placed just at the entrance of the meatus and completely filled with gauze, continuous with the packing of the wound. It serves the double purpose of giving a better contour to the meatal edge, and of preventing the packing becoming so adherent to the edge of the wound.

While in the exceptional case very nearly if not quite as rapid healing takes place in the non skin-grafted cavity, this result is by no means uniform. With a reasonably small

cavity, and flaps so placed from the canal as to advantageously assist in the spread of new epidermis, a favorable result is almost assured without further assistance. This is quite in accord with the teachings of general surgery; but one must not expect the skin to bridge a gap of more than half an inch.

With a large cavity, it would seem to be only reasonable to expect that a graft might render valuable assistance in shortening the treatment. Indeed, the brilliant results which followed the use of some of the first Thiersch grafts for this purpose led many operators to adopt this method in all cases. Some always apply the graft at once, others as invariably await a later stage, commonly from the fourth to the tenth day. One operator advises further curettement of the cavity at this time, as a more certain means of encouraging the graft to take.

That some, at least, of this abundance of detail is needless, is attested by the results in certain cases, which from neglect have disappeared from observation almost immediately after operation and without skin graft, only to reappear after months or years with a completely epidermatized cavity.

It is thought by many, however, that a suitable graft, successfully placed, lessens the period of convalescence decidedly. The chief objection advanced against its employment seems to rest on the fact that some operators experience a difficulty in having it "take".

Due care in the preparation of the cavity, however, will materially aid in accomplishing a successful result. If, under the best of conditions, failures occur, something is wrong with the technique. In this, the writer speaks not alone from his own experience, but from that of his colleagues as well. In the hands of several operators at our hospital the results are practically uniform.

No attempt is made at the present time to apply separate small grafts. A large thin graft two inches or more square is cut from the thigh with an ordinary amputating knife. The skin is immediately spread out on the place from which

it was cut, exactly as if it had not been severed from its attachments. A square of crepe de lise is then laid on it, and the skin and crepe together removed from the thigh. This simple device absolutely prevents rolling or curling of the edges. A piece of ordinary gauze, or rather several layers, which have previously been rolled into a cone, are then placed under one corner of the crepe and skin, in such a manner that the entire apex of the cone and at least one side is completely covered with skin the raw side out. One side of the cone is purposely left free of skin, as this side goes into apposition with the anterior wall of the canal, which is already covered with epidermis. The cone, or plug, is then introduced through the meatus, the point guided toward the middle ear, and then the gauze of the plug gradually fed into the cavity, expanding and completely filling it.

It must be remembered that the graft is adherent to a thin, smooth, silky crepe, and the pressure of the mass of gauze causes this to reach every part of the cavity, even where the meatus is much smaller than the inside, the gauze expanding in much the way that a bristle probang does. This gauze is allowed to remain undisturbed three or four days, at the end of which it is gently removed. The crepe de lise comes with it, but the skin, never. In the interval, it has become firmly attached to all denuded surface. There are naturally some loose ends where the graft is superimposed upon the epidermis of the meatus, and there are necessarily reduplications or folds in the graft, as it is impossible to adapt a flat surface to the inside of a sphere without such. These loose pieces finally slough out, and during the process there is a very active suppuration to deal with. In most of the cases the superficial layers of graft are cast off, but underneath there is the firm pink appearance of new skin, rapidly paling out. Immediately following the removal of the gauze plug, it is possible to demonstrate that the graft has "taken" by pulling upon it. It stands a very considerable strain, and if we succeed in detaching a piece, it causes bleeding, showing that it is definitely attached to its new resting place.

This method of applying the graft was described by the writer several years ago, and has been in use continually since. It appears to be simpler in execution than the Ballance method, and quite as certain of a successful result. The graft may be applied either at the primary operation or at a later period.

Following the throwing off of the superficial layers of epithelium a simple cleansing of the cavity with hydrogen peroxid may be permitted, but aside from keeping out the products of suppuration the less treatment given the better. The writer does not favor the use of drying powders, scarlet salve, or other medications, as they are apt to produce crusting and deceive one as to the true condition of the cavity. Under no conditions should packing of the cavity be continued.

Cases where the skin does not "take" pretty generally throughout the cavity are rare; but if such occur, and granulations form, they should be kept down by the free use of caustics. This is substantially the treatment that must be followed if no skin graft be used.

In summing up the record of progress, emphasis should be laid on the fact that while once the radical mastoid operation was looked upon as too severe a procedure to which to submit our private patients, it is now so treated that recovery takes place in as short a space of time as in the acute mastoid; and that while once the dressings were painful, protracted, and called for the exercise of as much skill as the operation itself, now, thanks to our modified technique, they are practically painless, and every contingency has been so carefully guarded against in the operation that a minimum amount of after care is required.

REFERENCES.

Ballance. Transactions Ninth International Otological Congress 1912.
Freeman. Skin Grafting 1912.
Hammond. Jour. A. M. A. Nov. 17th, 1906.

Discussion.

Dr. Frederick L. Jack, of Boston, Mass.—I have had the opportunity of following a few of the cases reported by Dr. Hammond, and can bear witness to the good results obtained by him. I differ with Dr. Hammond, however, upon certain small points. The metal clamps seem to me often to cause greater pain and more scars than the usual method of suturing. It is not usual to remove all of the facial ridge, yet Dr. Hammond has had no facial paralysis. The rubber tubing which he used for keeping the meatus in position is excellent, but I find the iodoform wick useful. Nothing was said about treatment of the Eustachian tube; presumably this was taken for granted. At times there is a moist spot at the orifice of the tube. I prefer Ballance's method of applying the graft when practical.

Dr. Edward B. Dench, of New York City, endorsed the metal clamps. He had given up sutures exclusively. He agreed with Dr. Jack in giving preference to the Ballance method rather than to that of Dr. Hammond. By means of a pipette it was possible to apply the graft to every nook and corner of the cavity more satisfactorily than with gauze. It was not necessary to interfere with the mucous membrane of the lumen in order to close the mouth of the tube. It was important to apply the graft over the mouth of the Eustachian tube, and for that reason the pipette possessed many advantages over the method proposed by Dr. Hammond. He advocated the primary graft, the secondary graft being useful only where the suppurative process cannot be entirely eradicated at the time of the operation, and where further operative interference will evidently be necessary.

Dr. Wendell C. Phillips, of New York City, had no objection to the use of skin grafting in the radical mastoid operation, but he did not consider it necessary. So long as he could get as good results as those reported he saw no

reason for skin grafting. He hoped he was not misquoting his fellow-workers at the Manhattan Eye and Ear Hospital, but he had not seen one used there for three or four years, and yet good results were obtained.

Dr. B. Alexander Randall, of Philadelphia, emphasized the removal of the cartilage from any flap that is made. He had taken that subject up before the Society fourteen years ago, urging the splitting of the flap into layers, which he had done ever since with satisfaction. Success with material which is already there emphasized the needlessness, in his hands of grafting Thiersch flaps. He did not eviscerate more than was absolutely needful; he did not believe the whole mastoid should be destroyed. When the canal lining had been turned out it was perfectly possible to split it, forming a flap of the periosteum, cutting it above and turning it down, then to dissect out the layer of cartilage, and then to loosen and turn upward the skin flap. To transplant a flap containing cartilage, as was usually advised was an essential mistake: it ruined the skin flap and the periosteal flap. He always transferred the skin flap to the upper part of the cavity, in proximity to the stapes, as this gave it freer movement, and the part of the cavity which was not covered would be quickly become covered. He did not feel that he had ever left undone anything which he should have done when he did not use a Thiersch skin graft.

Dr. George A. Leland, of Boston, Mass., described, and illustrated by blackboard drawings, the method employed by him for obliterating the posterior cavity in the radical mastoid operation, at the same time leaving the external meatus only a little larger than the original one. This is done by taking a periosteal flap, just below but up to the temporal ridge, its posterior edge being about 1½ to 2 inches behind the posterior meatal wall, its inferior edge beginning nearly at the tip of the mastoid process and extending obliquely upward and backward to meet the rounded posterior tip; the thickness, including the fascia, periosteum

and all tissues between, varies very much. The excavation is made after elevation of this flap, and then it is folded inward and its posterior tip packed into the antrum, and its posterior edge along the excavation depression behind the facial ridge. The external meatus, having been incised above and below posteriorly and dilated sufficiently to admit the end of the little or ring finger, is kept thus open by the insertion of a stiff rubber tube, the tongue formed by the posterior wall of the external canal, deprived of the inner part of its cartilage, is pressed upon by the rubber tube, and the flap presses against this tongue and is kept there several days (8 or 9) by small wads of gauze which hold the flap in place in the depth of the wound. The original curvilinear incision is then sutured above and below, leaving ¾ to 1 inch open, through which light packing, to keep the lips open, is inserted, and through which in eight or nine days the anchoring wads are carefully removed. The cavity can then be lightly packed or powdered with boracic acid or iodoform, and the rubber tube is kept drained by thin gauze wicks and through it the fundus can be easily treated, kept dry, and granulations kept down, dermatization taking place in a comparatively short time.

Dr. Eugene A. Crockett, of Boston, Mass., said his experience with the radical operation covered several methods. He began by using the primary graft for several years, then for five or six years he used the secondary graft. He then came to the conclusion that it was not best to use any graft at all, but to do the usual operation, wiping dry afterward twice a day and powdering with boric acid. For six or seven years he had used the last method and had found healing to take place more satisfactorily than with the flap. The hearing was as good. He cited the case of a girl, twenty-three years of age, who presented the symptoms of leptomeningitis. He operated upon her with no one to assist, twenty-seven miles out of Boston. He did not use a graft, but packed and used a Koerner flap. He

went out to see her on the fourth, seventh and tenth days. The temperature came down after the third day, then it rose again and the packing was taken out on the fourth day on account of the temperatures. Healing took place in twenty-six days. If he had used grafting he would have required elaborate paraphernalia, trained assistants, etc. Cases of this sort could be handled in a simple way. Anyone who would visit the Massachusetts Charitable Eye and Ear Infirmary would be convinced, he thought, that the granulation method is the best.

Dr. Ewing W. Day, of Pittsburg, Pa., took exception to what was said concerning the use of adrenalin. In the radical mastoid operation, under local or general anesthesia, he had never seen any devitalization of tissue from its use. It shortened the operation from twenty to twenty-five minutes. After once abandoning skin-grafts, he now used it in the majority of cases. His associate, Dr. Waldie, would not do a radical mastoid operation without skin grafting, holdng that recovery is quicker and results better.

Dr. Hammond, in closing the discussion, emphasized the fact that the paper dwelt with after-treatment, for which reason he did not discuss the method of clearing out the Eustachian tube, taking out the facial ridge, and other points of the radical mastoid operation. He believed one gets far better results if one takes the time to thoroughly clear out the temporal bone. One who knows the anatomy had nothing to fear concerning injury to the facial nerve. The cavity was large and symmetrical and it was easy to graft it. Regarding the entire question of grafting the mastoid, for a number of years he had advocated grafting, and he had seen such brilliant results with it that he had come to consider it as necessary; now, however, he was willing to concede that certain suitable cases did well without the graft, although healing was not so rapid as where the graft was employed. He had not seen injuries resulting to the hearing following the graft, and would take exception to

the statement that there is more dermoid proliferation after grafting. Referring to Dr. Randall's remarks concerning the thickness of the graft taken from the thigh and the undesirability of using it in the region of the tympanic cavity because of its interference with hearing, he said the graft which he uses is so thin that one can almost read a newspaper through it. This method was being successfully used by many of his colleagues.

INFLUENZAL OTITIS.

By S. MacCUEN SMITH, M. D., Philadelphia, Pa.

The subject of aural disease complicating influenza, more especially the various manifestations, or rather, the lack of manifestations during an influenzal attack, seems to be of sufficient importance to warrant a few observations on this very common disease.

Were it not for the small caliber of the Eustachian tube and the direction of the movements of the ciliated epithelium lining the same, we would have a very much larger percentage of ear diseases complicating the various abnormalities of the naso-pharynx. This functional activity of the tubal epithelium, the direction of which is from the tympanic cavity to the naso-pharynx, can be likened somewhat to that of the downward peristaltic action of the bowels.

The fact that the tube is relatively much larger in infancy and early childhood, however, accounts for the ease with which the various microörganisms can overcome the resistance of the expulsive action of the ciliated epithelium during violent coughing, vomiting, sneezing and blowing the nose, and the consequent greater prevalence of aural suppurations in early life.

Then, again, in debilitating diseases, such as tuberculosis, pneumonia, typhoid fever and certain types of influenza, the caliber of the tube is considerably increased by shrinkage or atrophy of its mucosa. This no doubt accounts for the fact that the majority of aural suppurations occur during the latter part of, or convalescence from, various diseases, the bacteria the more readily entering the tympanic cavity because of the impaired resistance of the tubal epithelium. Furthermore, bacteria enter the tympanic or mastoid cavity through the blood vessels and lymph channels. This would seem to be the logical explanation for the development of

so-called primary tympanic and mastoid inflammation, more especially the latter. Indeed we have no other means of explaining such conditions complicating general infections, particularly in the absence of any trace of naso-pharyngeal disease.

Although the claim may be justified that it is virtually impossible to get a pure mono-bacterial culture from an acute suppurative otitis media at the time of incision of the membrana tympani, nevertheless, from a clinical viewpoint, the results of such a microscopical examination can be accepted as definitely defining the character of bacterial invasion. If, therefore, under such circumstances we find the influenza bacillus, I am inclined to accept the infection as being of this particular type, though the patient may not present marked general symptoms of influenza.

In a goodly number of cases, more especially encountered during this past winter, we have found extensive aural complications in influenza, although the patients were not sufficiently ill to be confined to their beds. Then, again, in another type of the disease, our patients have been systemically seriously ill, whereas the aural symptoms have been decidedly mild in character.

I am well aware of the tendency of the medical profession, in the present day, to call almost any ailment influenza, in cases where no other disease definitely manifests itself, and this unscientific designation of an illness is analogous to a former period when all doubtful diseases were supposed to be malaria, yet I feel, as above stated, that a culture taken directly from the tympanic cavity does enable us to state definitely whether the otitic involvement is influenzal or otherwise, keeping in mind, of course, that the discharge becomes mixed, or polybacterial, in character very shortly after exposure to the air.

We must give due consideration to, and recognize the formidable importance of the influenza bacillus, when this particular microörganism invades the human economy. For example, I quote the published findings of Lord, who, "in a series of 186 specimens of sputum from cases unselected

except to exclude those with tubercle bacilli, found organisms resembling influenza bacilli in 50% and in almost pure culture in 25%. In eight patients, their constant presence in the sputum was demonstrated for two and one-half years, and the suspicion is strong that they had been present for a much longer time.'' This observation was made during an inter-epidemic period, and illustrates the endurance and vitality of the influenza bacilli and their consequent power for almost indefinite destruction. Furthermore, the results of this scientific investigation furnish in detail a conspicuous example of the insidious nature of this organism, in some instances, in striking contrast to its virulence in others.

The aural destruction in some of my influenzal cases has been so rapid that we would naturally suspect that the streptococcus must have been an important factor. However, we were unable to find any microörganisms except that of influenza in at least three cases, in each of which the bony destruction was very marked, causing exposure of the sinus and dura all in the space of about twenty-four hours, and this without any suffering from pain or other general manifestations on the part of the patient. In one of these cases a large sequestrum, involving the greater part of the tip, was removed, this patient giving the early symptoms of Bezold's mastoiditis during the preparation for the operation.

I may be permitted to digress for a moment from the subject of influenzal infection in order to state that, in my experience, pneumococcic otitis is very similar to the influenzal form in showing the extreme degree of destruction peculiar to the latter, as well as the tendency towards metastasis.

While the usual case of influenzal invasion is characterized by severe pain, decided rise of temperature, depression and exhaustion, I wish to emphasize that type of case mentioned above, in which the destruction occurs very rapidly and the complications develop almost simultaneously, without the symptoms above enumerated. This leads us again to lay stress upon the importance of the well-recognized

procedure of early incision of the membrane for the evacuation of tympanic secretion, as well as immediate major surgical intervention if this more simple measure does not give prompt relief.

So long as influenzal invasion attacks the young or those with a thin and more or less porous cortex, the direction of the inflammatory process is usually external, as is commonly manifested by the characteristic pain and swelling over the mastoid. In those cases, however, where the mastoid is small and the cortex comparatively thick and dense, the course of erosion will naturally follow the path of least resistance and invade the sinus or interior of the skull. As it is clearly impossible for us to definitely determine this point in many of these rapidly developing infectious diseases, the safety of our patient, I wish again to repeat, lies in early operative intervention.

The first case that I wish to report is that of H. C., male, 7 years of age. The parents claim that prior to the present attack, this child never suffered from any form of ear disease whatsoever. Three days before his admission to the hospital, the boy complained of a slight pain in the right ear, not sufficient, however, to interfere with his rest that night. The following day the pain recurred and the attending physician ordered a hot water bag applied to the ear. The patient also slept well this second night. On the afternoon of the following day, through a spontaneous rupture of the drumhead, a slight discharge appeared. Co-incident with this, or forty-eight hours after the initial pain, the parents noticed a swelling over the mastoid process, extending below the tip into the neck. The next day, or three days after the inset of the disease, the boy was brought to the Jefferson Hospital and operated upon at noon.

On examination we found the external auditory canal well filled with a creamy yellow pus, without odor. There was considerable edema and swelling throughout the entire osseous canal. The opening in the drumhead occupied the greater part of the posterior half. On operation, we found the edema over the mastoid process to be about three-fourths

of an inch thick. There were two small carious openings through the cortex, which bled freely as the periosteum was separated. The entire cortex was thin, and upon its removal we found the sinus very far forward, superficial, and exposed through carious erosion in the superior half. The removal of some additional bone over the sinus provided for the escape of a considerable quantity of pus, and we therefore uncovered the vessel from the knee to the bulb. The sinus was bathed in such a large quantity of free pus, which escaped on the removal of the remaining bone covering the same, that we suspected a possible thrombus, which might have become disintegrated, the pus escaping from the bulb. As we approached the neck, however, we discovered that the pus had burrowed through the tip and we were evacuating the same from a Bezold's variety of mastoiditis. We therefore concluded not to interfere with the sinus for at least twenty-four hours, or a sufficient length of time to enable us to determine whether the child was actually suffering from a sinus thrombosis, inasmuch as there was no evidence, by palpation or systemic manifestations, that a clot had formed within the vessel. The antrum was also well filled with a quantity of pus and some granulation tissue.

The patient made a slow but uninterrupted recovery, returning home at the end of the fourth week following operation. This case well illustrates the rapid destruction that frequently accompanies an influenzal otitic inflammation, and also *forcibly* illustrates the almost total absence of suffering by the patient.

It may be well, also, in this connection, to call attention to the fact that even though an early myringotomy had been done, in this particular instance, it is doubtful whether the rapid destruction could have been prevented or even modified, for it would seem, in accordance with the findings, as stated above, that the mastoid process must have been involved simultaneously with the tympanic cavity.

Not long since I operated on a case showing no general symptoms, but presenting equally destructive changes, in which the middle ear gave no evidence of inflammation other

than a mild degree of myringitis. In this latter case we probably would be justified in claiming a primary mastoiditis, and yet, as stated on former occasions, I believe that the infection originated in the tympanic cavity and that the mastoid was secondarily involved, the former, on account of the patulous Eustachian tube, being able to drain and recover itself.

The second case is that of H. J. K., male, aged 54 years, apparently in robust health. Three days prior to admittance to the Jefferson Hospital, this patient suffered from an attack of influenza, more especially involving the bronchi, at the end of which time he complained of pain in the right ear. The membrana tympani was decidedly red, though not bulging, but on the following day was markedly bulging and the sero-sanguineous fluid was evacuated by a prompt incision. The aural pain was relieved for about thirty-six hours, the ear in the meantime discharging freely. At the end of this period the patient complained of discomfort in the occipital region, as well as a return of the aural pain, all of which continued during the succeeding six or seven days. In the interval he also complained of pain over the mastoid process, which was increased on pressure.

During the following twenty-four hours the ear discharged profusely and the pain in the back of the head and over the mastoid process also markedly increased, although the patient's temperature and pulse remained practically normal. On this day, or eight days from the time of his initial pain, there was, in addition to the profuse aural discharge, a marked drooping of the superior and posterior wall. In addition, he became more or less erratic and irritable, the tenderness over the mastoid process was more pronounced, and we performed a simple mastoid operation. On dividing the soft parts, the cortex bled freely and there was a small carious erosion through which free pus escaped, apparently under pressure. On removing the cortex, we found the cavity to be well filled with pus and granulation tissue. There was a perisinus abscess formation exposing

the greater part of the vessel, and also carious erosion exposing the dura, through the tegmen antri.

Although the patient's temperature and pulse remained virtually normal, his mental state and general head discomfort continued to grow progressively worse. Three days after the mastoid operation the temperature suddenly jumped to 102.3°, and the pulse to 112, and the polymorphonuclear percentage, which was 89, with a leukocytosis of 19,800, suddenly rose to 94, with a leukocytosis of 20,200. Accordingly, we promptly opened the wound, incised the exposed dura, and evacuated a temporo-sphenoidal abscess formation. This immediately relieved the breathing, which had been very labored, the patient requiring an additional amount of ether for the completion of the operation. During the first part of the following day his general improvement, including the mental state, was very marked, though he died at the end of about twenty-one hours, from acute pulmonary edema. As usual, we were unable to secure an autopsy and therefore I regret my inability to report on the exact pathological findings.

Five years previously, this same patient suffered from a suppurative influenzal otitis of the opposite ear, which progressed so rapidly and caused so much necrotic erosion that it was necessary to do a radical operation within the short space of about one week. This case well illustrates not only the rapid osseous destruction of an influenzal otitis, but also shows the rapidity with which a brain abscess formation can develop under the destructive influence of this particular type of infection.

Discussion.

Dr. Stephen H. Lutz, of Brooklyn, N. Y., referred to a series of examinations, about a year ago, to determine how many cases of so-called "grippe," out of one hundred examined, were influenza. Two were true influenza, the rest were due to various infections. The influenza bacillus was very hard to grow in the ordinary Board of Health tubes which

are furnished for diphtheria. It was such a delicate organism that it grew easily only on special medium. Dr. Smith found the influenza germ, evidently having encountered an epidemic. He wished to protest against the term "grippe." It was often stated that these cases had, in the beginning, the grippe, whereas the majority of them have the ordinary infections so often encountered, most often staphylococcus aurens, pneumococcus or streptococcus of some variety.

A BRIEF CONSIDERATION OF CERTAIN RECENT VIEWS REGARDING OTOSCLEROSIS.

By THOMAS J. HARRIS, New York, N. Y.

The study of that form of progressive hard-hearing known under a variety of names, but most generally referred to as otosclerosis, is frought with peculiar and to a large degree uncontrollable difficulties. The affection is not like that of adenoids, something that can be gotten at and easily removed for examination. For accurate investigation, the microscopic examination of the temporal bones of those in whom the disease has been diagnosed during life, is essential. The obtaining of these is difficult anywhere, as there is nothing, per se, in the disease which is wont to occur in the young, to hasten the death of the person affected, and especially difficult in this country where post-mortem material is so exceedingly hard to secure. An equally essential requirement, possessed by few otologists, is that of being well grounded in pathology. As a result, the total number of cases investigated is not large and these are almost entirely limited to those studied by our German confreres.

It is with no claim to any expert pathologic knowledge, that I have presumed to present another paper on this subject, but rather that as a result of some personal study and of an opportunity recently to meet a number of those who have given especial attention to it, I wish in a judicial way to weigh and consider briefly the various views which have been advanced regarding the nature of this disease. I am fully aware that in the minds of many of you the subject appears hopeless of solution. Indeed, in one of the best short papers on the subject which has recently appeared, that from the pen of a member of this society, Dr. Sheppard, the pessimistic view held by certain foreign writers was advanced, i. e., that the diagnosis even of otosclerosis is impossible.

For a proper consideration of the subject, a brief reference to its history is necessary. The number of investigators is

few, and the total number of cases, microscopically studied, collected by Denker up to 1904 is quite limited. The term otosclerosis, as you are aware, was first used by Joseph Toynbee in 1857 in his descriptive catalogue of 1,149 specimens, where he describes 126 cases of union of the stapes in the oval window. This term was used for many years to describe such an obstruction to the sound waves, whether of a membraneous or bony nature. But it was not until 1885 that it was demonstrated microscopically and physically by Bezold, that the loss of hearing for low sounds was the result of such rigidity in the oval window. The first microscopic examination of a diseased temporal bone in a case of otosclerosis was made by Katz 1890. He was followed by Habermann who, in 1892, reported his first case. In 1893, Bezold and Scheibe reported their first case and in the same year Politzer in this country, and if we are not mistaken, before this Society, reported 16 cases, all observed by him during life, and studied histologically post-mortem. In 1894, Bezold reported his second case; in 1898, Siebenmann reported four cases. In 1901, Scheibe reported two cases, in the same year Habermann reported a second case, and Katz a second case, while in 1903, Habermann reported ten temporal bones observed in six patients, a total of 34 cases. Since then Panse has reported two cases, Jörgen Möller one case, Brühl five cases, Schilling one case, Otto Mayer one case, and most recently Manasse has described seventeen temporal bones in ten patients.

The early view in regard to the etiology of the affection, was that it proceeded from a disease of the middle ear. This is undoubtedly correct in the cases of Katz, where both from the history and clinical picture, a previous chronic suppuration of the middle ear was evident. In those of Scheibe and of the ten cases of Habermann there is likewise a clear history of middle ear disease. Politzer was the first one to advance the view, which he still maintains, that the disease is a primary affection of the labyrinthine capsule, originating not in the periosteum but in the bone itself. As a result, new bone tissue is developed which gradually presses out the old bone and advances to the oval window and the stapes, leading finally to

a complete stapes anchylosis. Siebenmann was the first to make a careful study of the affection and he is of the opinion that the spongifying process does not proceed from the periosteum, nor yet does it develop as a primary affection of of the bone. From the cases which he studied, he is of the opinion that the starting point is to be found on the border between the labyrinthine capsule, which is primarily developed out of cartilage, and the connective tissue bone (Bindegewebesknochen) secondarily developed from the periosteum, and that the earliest stage of the disease is to be found in a lacunary resorption of the bone by means of the Haversian canals which become dilated into large lymph spaces, although at the same time in other areas there is apposition going on by means of osteoblasts. The spongification in his opinion is the last stage of a growth which does not normally occur in the temporal bone, but is the rule in other bones, and follows here the entire disappearance of the cartilage present in the intercellular spaces and on the border of the oval and round windows and also by the growth of compact bone into the osteoid tissue, and finally into the spongified tissue.

Habermann, who studied the subject with equal care, but with specimens derived from patients who had suffered from middle ear disease, believes that the affection proceeds along the blood vessels from the middle ear into the inner ear. Finally, Denker, as a result of a study of the collected literature as well as from personal investigation, in 1904 reached the conclusion that the disease might be in some cases secondary to middle ear disease, but was usually of a primary nature, and recognized at that time the difficulties which have since presented themselves, due to the variable location in the labyrinthine capsule of the diseased areas. Since Denker's monograph on the subject, the views of investigators have been materially altered as the result of a masterly study of the subject by Manasse, of Strassburg, entitled "Die Ostitis Chronica Metaplastica der Menschliche Labyrinthkapsel." Manasse previously in Basle advanced, but only last year published this result of his studies in the examination of 17 temporal bones derived from ten cases, a study extending over

twenty years. As in the case of Siebenmann, the contributions of Manasse are particularly important because of the fact that he is a trained pathologist.

The views of Manasse it will be necessary to state somewhat particularly.

1. In the first place he confirms the observations of Siebenmann and Habermann, that the predilection area for the diseased process is the anterior border of the oval window. This, however, as they have also pointed out, is not always the case. Three times the porus acusticus internus was involved, twice the round window, three times the apex of the cochlea, and once the anterior portion of the labyrinthine capsule and the posterior semi-circular canal.

2. Of particular interest are Manasse's views regarding the histology of this bone affection. He believes that the "disease is virtually a transformation of the labyrinthine capsule. The compact bone is to a greater or less extent replaced by new (foreign) bone substance, which is everywhere sharply contrasted with the old bone tissue. The new bone differs in its structure according to the age in which one finds it. The chief difference as compared with the old bone, is that instead of compact bone, a system of bony trabeculae is to be seen between which there lies a greater or less number of large open spaces. These spaces are composed in the younger parts of a tissue very rich in cells, which in the older parts becomes fibrous and at times is replaced by fat tissue or rarely even by Schleimgewebe. The entire newly formed tissue would be spoken of in general as *spongifying bone*. Later the open spaces become smaller. They contain, however, very little of real marrow, only blood vessels. The bone becomes much harder and denser, and develops a regular lamellae system so that without doubt it can be regarded as compact bone, and can pass very rarely into firm sclerotic bone entirely lacking in marrow and with few blood vessels."

Again quoting Manasse, "With few exceptions, two forms of bone will be met with in the diseased areas, both representing in the majority of cases the early stage of the

disease. First, small open spaces with pronounced bony trabeculae, coloring very bright with hamatoxlyn, and second, larger open spaces taking a darker stain and with fewer and smaller areas of bone between. The bright are the older diseased areas; the dark, the newer. Neither is composed of compact bone and as Siebenmann pointed out, both are lacking in the intercellular spaces containing cartilage, which are characteristic of the normal labyrinthine capsule. The newer bone tissue shows between the thin, bony trabeculae large open spaces containing a tissue rich in round cells, cylindrical cells, and osteoblasts as well as osteoclasts. There are no large blood vessels, only capillaries, no compact connective tissue, no myeoblasts nor red blood corpuscles, no fat tissue, and only a small amount of intercellular substance. The picture as a whole is one of granulation or new connective tissue, not of lymphoid marrow. Certain spots in the bone substance which take a deep blue color, are transparent, contain only small three-cornered bone cells such as is met with in immature bone, and show a peculiar *sievelike rarefaction*, the round, long or jagged holes of which are each filled out by a single cell. These holes often come together, forming canals. They also contain capillaries and empty into the larger open spaces. Osteoblasts and osteoclasts are never met with in the narrow spaces of these deeply colored bones. The older form of the new bone is composed of larger bony trabeculae which take a brilliant red color and which form a distinct network. The bony substance shows both jagged and plump bone corpuscles, but no lamellae system. The open spaces contain few cells, often only one large, wide, thin-walled blood vessel, but also a fairly soft connective tissue with lymphocytes, spindle or star-shaped cells, and several vessels. This fibrous marrow passes occasionally over into the fat marrow, and at times into the yellow marrow. Osteoclasts are met with more often in the larger areas than in the smaller. In the older parts of the large areas, the picture is one usually met with where the bone has been forced out of Haversian

canals, but nowhere is there a reforming of the intercellular spaces containing cartilage.

We cannot go too deeply into the minute histology represented in Manasse's studies, but the question of the primary source of the diseased process must be referred to. Manasse thought for a long time that this was to be found in the osteoclasts which are seen in the perivascular connective tissue in the bone, but his more careful study showed him that the initial stage proceeds from the blood vessels themselves. The second interesting question is the origin of the sievelike rarefaction which represents the most recent stages of the new bony growth. These minute openings, Manasse believes to be where the bone corpuscles were previously found. As he remarks, this form of bone absorption is not unknown. M. B. Schmidt, among others, has carefully described this process. Manasse's conclusions are as follows:—"The alteration of the labyrinthine capsule begins with the formation of osteoid and granulation tissue within the preformed blood vessel spaces. The old bone is destroyed as the result of this new tissue growth by the simple process of compression, not through lacunary absorption, and the new bone tissue takes its place. This has only an intermediate character, in so far as it in turn is destroyed by two different methods of absorption. First, through a sievelike rarefaction of the new bone tissue, and second through lacunary absorption with the formation of osteoclasts. There follows a third deposit of bone in the marrow spaces as the result of osteoblast activity, which is at first spongified in character, and which does not disappear. As time goes on, it becomes denser and harder, and finally fibrous. The marrow spaces become smaller, the bony trabeculae always thicker, a lamellae system develops, and finally compact bone is formed which is always to be sharply differentiated from the old labyrinthine tissue. The whole process is to Manasse's mind an inflammatory one, and he has accordingly named it as just noted 'ostitis chronica metaplastica der labyrinthkapsel.'"

With these views most recent investigators agree, including Otto Mayer, Panse in his newly appeared "Pathology of the

Ear," and Brühl, except that Brühl is not prepared to admit that the process is a true inflammation. Manasse does not, however, stop here, but in addition to the changes in the bone, describes changes in the membranous layrinth and claims based on his 17 specimens where such changes without a single exception are met with, that alterations in one or more portions of the sound perceiving apparatus, namely, either atrophy in the organ of Corti, connective tissue deposit in the fine nerve filaments in the cochlea, similar changes in the spiral ganglion, or finally in the auditory nerve itself, are an essential part of the disease. He contends, indeed, that in every case of chronic hard-hearing of a high degree, whatever the cause may be, there are always the same changes in the membranous labyrinth. He does not presume at the present time to say whether the bone changes cause those in the labyrinth, or vice versa, although in one case of his where the labyrinth was diseased on both sides, but the patient heard better in the ear where the bone was not affected, he does not think the labyrinth could have been involved as the result of the bone disease.

From these views of Manasse regarding the membranous labyrinth, Brühl vigorously, and it seems to us with good reason, disagrees, or rather argues "not proven." In the first place, nothing is more difficult to determine than what changes in the membranous labyrinth are pathologic and what are post-mortem. The tissues are exceedingly delicate, and subject to post-mortem influences in the time elapsing after death, and from the processes adopted for their examination. This objection Manasse admits and defines certain alterations found, as pathologic. Further, it is impossible to positively show whether the change in the soft parts does not develop long after the original bone disease, and even as a result of the intercurrent affections, possibly acute, which carried the patient off. Brühl indeed insists that such changes are in no way characteristic as they are met with in all severe affections of the middle ear. Finally, it is too much to say that either clinically or anatomically they are always present. In two young patients recently examined

by Brühl suffering from otosclerosis, the one 34 years of age, and the other 24, there was no lowering whatever of the upper tone limit, while in a case just reported by him showing microscopically the characteristic bone changes, the organ of Corti was entirely normal. The most that we can assert, then, at present, is that changes in the membranous labyrinth are frequently though not invariably present; whether they are a necessary part of the anatomic disease is yet unproven, but they are of sufficient importance to be always borne in mind. Functional tests showing nerve deafness do not necessarily prove that the cause lies in the disease under discussion, but if slow in onset and if all other causes can be excluded, render such alterations in the membranous labyrinth highly probable. What is urgently required in order to throw more light on this difficult question is the careful study of temporal bones of young subjects, in whom otosclerosis has been diagnosed clinically. This, as we said at the outset, is manifestly difficult to accomplish. The one objection to Manasse's studies is that his specimens are chiefly from aged people where we would expect normally, changes in the labyrinth.

Of far more interest to us as clinicians, than Manasse's histological studies, is his statement in regard to anchylosis of the stapes in his cases, and the deductions which he draws from this. In only three cases, five temporal bones, was anchylosis found. In the remaining twelve bones, the joint was uninvolved. Manasse concludes, based on these findings, that anatomically speaking, stapes anchylosis can no longer be regarded as an essential part of the disease, nor even can it properly be said that these findings are due to the early stage of the disease, and that later there would have been such an involvement, for in two cases at least, the microscope shows the process to have been evidently an old one, which had stopped short of the stapes and the annular ligament. Manasse argues, furthermore, that even if stapes anchylosis is met with, it is impossible to say that it is bony in nature, and due to the disease in question. He supports this contention by giving the histological findings

of three cases of anchylosis, the first due to a lime deposit, the second due to otitis media chronica adhesiva, and the third, a result of a general periostitis of the stapes and of the inner wall of the middle ear. It is evident that whatever the cause of the anchylosis, the functional tests will be the same, and Manasse accordingly advances the view mentioned previously and shared in by Panse, that chronic progressive hard-hearing is not possible of diagnosis. We can readily understand how with all that has been written to throw doubt on the subject, one can gain this pessimistic opinion. We cannot, however, admit Manasse's conclusion. It must not be forgotten that ostitis chronica metaplastica der labyrinthkapsel, so minutely and painstakingly described by him, is strictly an anatomic disease. It is undoubtedly true that it may develop in other parts of the labyrinth capsule than the oval window, and in such cases may produce no symptoms unless the membranous labyrinth is involved, when the upper notes will be affected. It is undoubtedly correct to say that there are many such cases which have never been recognized. It is, however, the clinical manifestations of the disease which interest us and alone concern us, and only when the disease has advanced to involving the stapes and causes an impairment or fixidity of that bone, we speak of it in a clinical sense. So it is proper to say that while there is an anatomic disease, there is also a clinical disease, and this, for a lack of a better name, will continue to be known under the name of *otosclerosis*.

While it is true that any form of anchylosis will produce the same functional tests, other causes than that due to a bone occlusion are so rare as to be excluded, although it must be borne in mind that such exceptions do occur. Panse states that he recalls a number of cases of deafness developing subsequent to confinement, which from the clinical course, the functional tests, the otologic picture, and the history of heredity, would have been regarded as otosclerosis. They, nevertheless, "by use of the catheter and massage, made a marked and lasting improvement." Such exceptions

undoubtedly occur, and are constantly to be borne in mind in pronouncing too promptly that the case is hopeless.

To sum up, we feel that the pessimism in regard to understanding and diagnosing this disease is not warranted, and that while it is true that much is yet to be learned both as regards the histology and the etiology, and that we can offer little for a cure so long as the latter is so obscure, we can confidently say that in the majority of cases a determination of the disease is entirely possible. While nine years have elapsed since Denker's book on the subject, we regard his description of the clinical disease an eminently correct one.

"In the cases of progressive hard-hearing, which show unchanged or virtually normal drum membrane, a patent eustachian tube and the Bezold triad of symptoms, we may conclude that the pathological changes are only in the stapes and the annular ligament and in the bony areas bordering on the oval window. In the other cases where the functional test does not give the Bezold triad, but there is a pronounced reduction of the upper hearing limit, bone conduction is not lengthened, and Rinne is not pronouncedly negative, there is in addition to the disease of the oval window an extension of the process further into the capsule of the labyrinth, or an involvement of the membranous labyrinth. While in most of the cases the mucous membrane of the middle ear shows no change, in the cases of Scheibe and Habermann, and in those of Katz, the cause undoubtedly lay in a chronic middle-ear suppuration. Consequently, as we took occasion in a former paper to point out, this or a similar middle-ear condition is not to be lost sight of in searching for a possible etiological factor.

1. *Plate 1—Fig. 1—Case 1.*

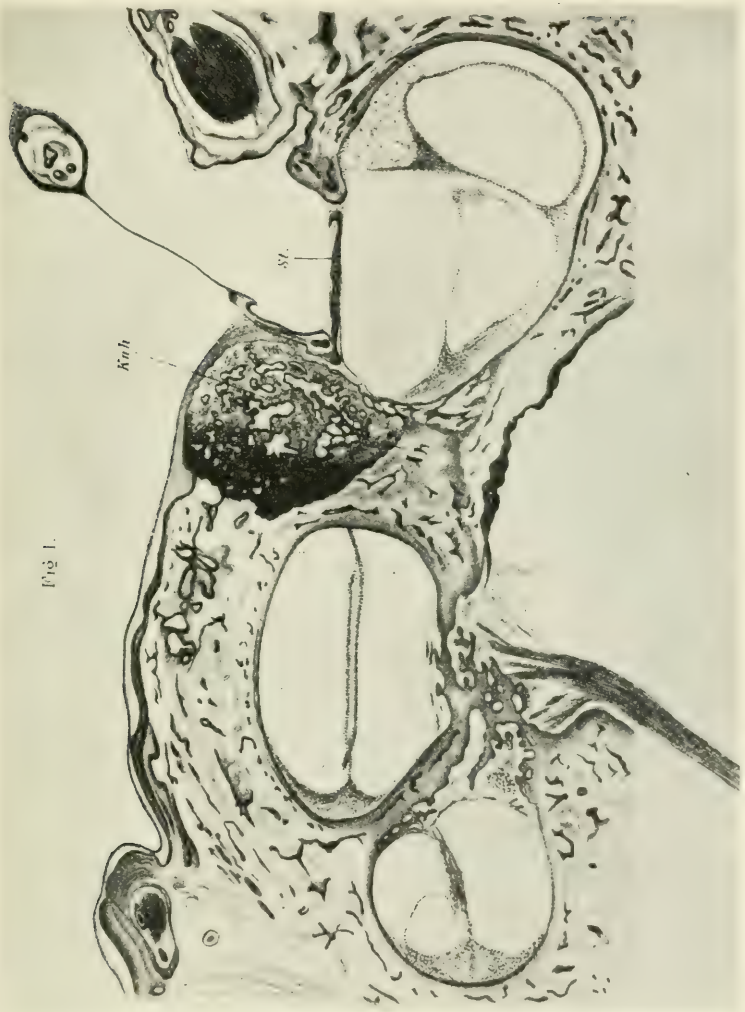


Fig 1.

Horizontal section through the region of the oval window. A small area of new formed bone on the anterior border of the oval window. The mass is light in color near the window, darker in color near the cochlea. The last are the recent, the first are the older portions. The mass extends somewhat into the window niche, but does not involve the stapes nor the annular ligament. There is no stapes anchylosis. In the cochlea can be seen atrophy of the organ of Corti, and an atrophy of the connective tissue growth in the spiral ganglion. The atrophy of the acoustic nerve is the result of the tumor which existed in this region. The cochlea, the sacculus and the utriculus are filled with hyalin. (From Manasse's collection with permission.)

2. *Plate 1—Fig. 1—Case 8.*

Advanced disease of the labyrinthine capsule. Almost the entire bony cochlea is transformed by the ostetic process. Only a small spot shows normal labyrinthine bone, N. L .K. We can see with this weak enlargement that the larger part of the new formed bone is very old, light in color, poor in blood vessels, and formed out of dense sclerotic bone tissue. Only on the inner periosteum can be seen a few dark colored recent portions. K. N. H. A small area of new formed bone. Complete stapes anchylosis, with the foot-plate strongly involved in the new bone formation, particularly on the posterior border. Degenerative atrophy in the membranous labyrinth, both portions of the acoustic nerve very thin. A. N. c. Atrophic cochlea nerve. A. N. b. Atrophic vestibular nerve. (From Manasse's collection with permission.)

3. *Plate 5—Fig. 5—Case 10. Right.*

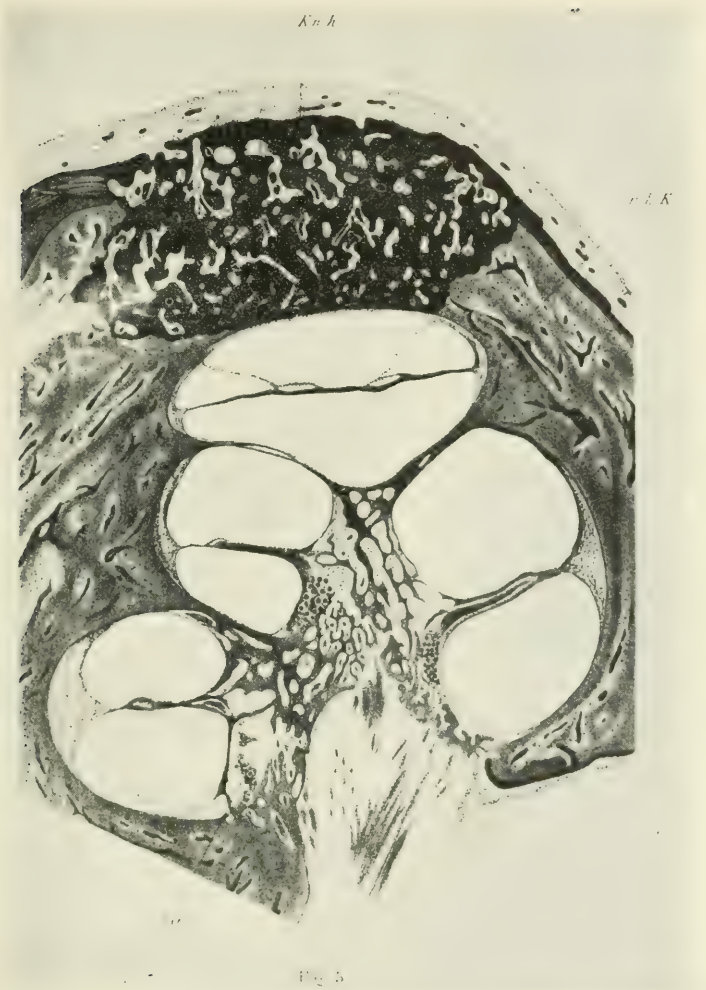
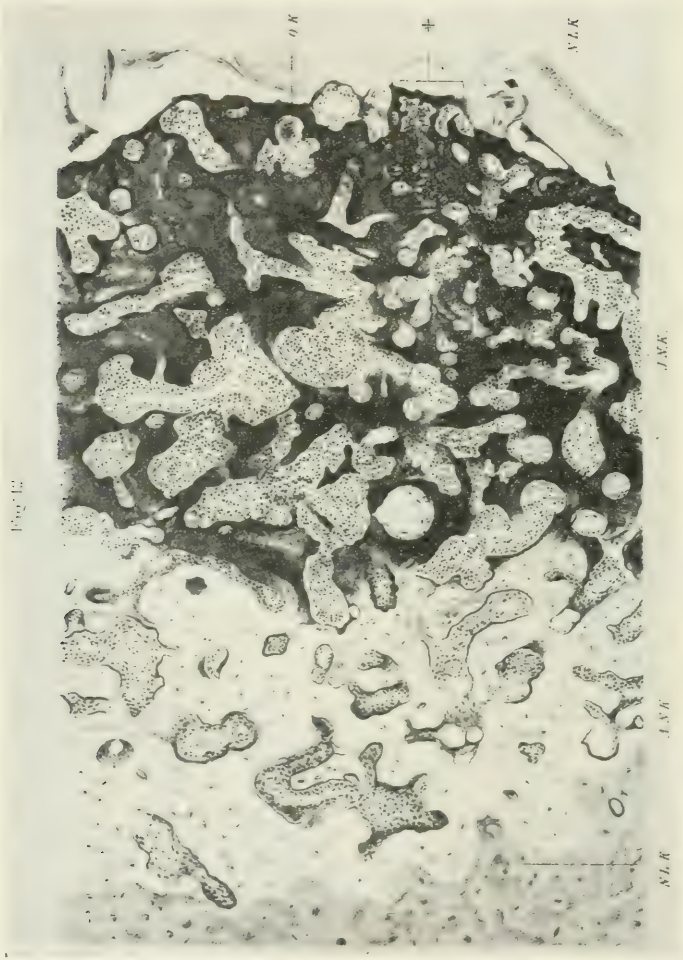


Fig. 5

A section through the cochlea; circumscribed pathological area of bone, (K. n. h.), in the capsule extending to the cochlea. The transformed bone involves the entire thickness of the capsule from the periosteum of the middle ear to the inner periosteum of the cochlea. It is formed almost exclusively of recent newly formed bone as can be seen from the dark color. In the membranous labyrinth, degenerative atrophy, the organ of Corti, the spiral ganglion and in the fine ramifications in the cochlea. The sinking of the Reissner's membrane is not pathological but post mortem. N. L. K. Normal labyrinthine bone. (From Manasse's collection with permission.)

4. *Plate 9—Fig. 12—Case 1.*



Section through the portion of the new formed bone lying near the cochlea. A stronger enlargement. We see on the left the light colored older portions of the new formed bone. A. N. K. On the right, the dark colored younger portions. J. N. K. Both are sharply separated from the normal labyrinthine bone. N. L. K. The former consists of dense bony tissue containing a few large vessels. The latter contains many more open spaces and many more cell element. At O. K. we see an osteoclast in the new formed bone. Nowhere are osteoclasts to be seen on the border of the pathological and the normal bone. Here always the newly formed bony substance crowds close on to the old labyrinthine bone without any cell elements. The star shows the cribiform rarefaction of the new bony growth. (From Manasse's collection with permission.)

5. *Plate 10—Fig. 13.*

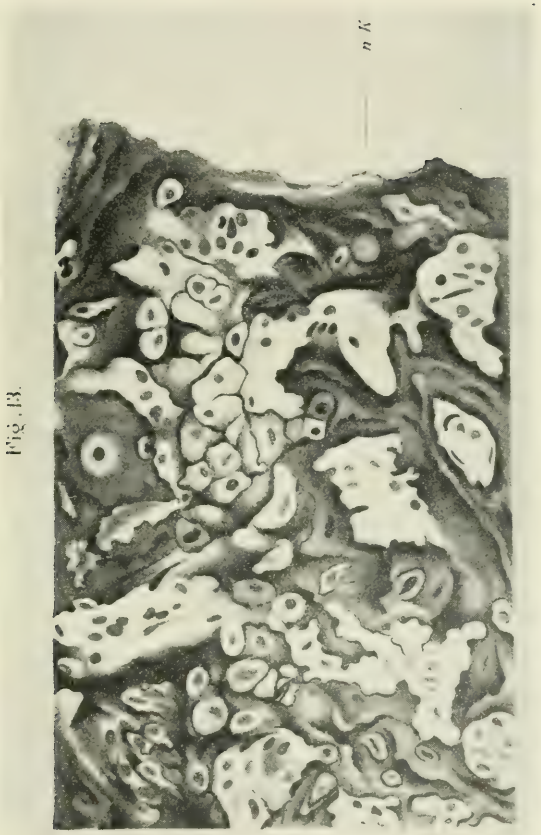
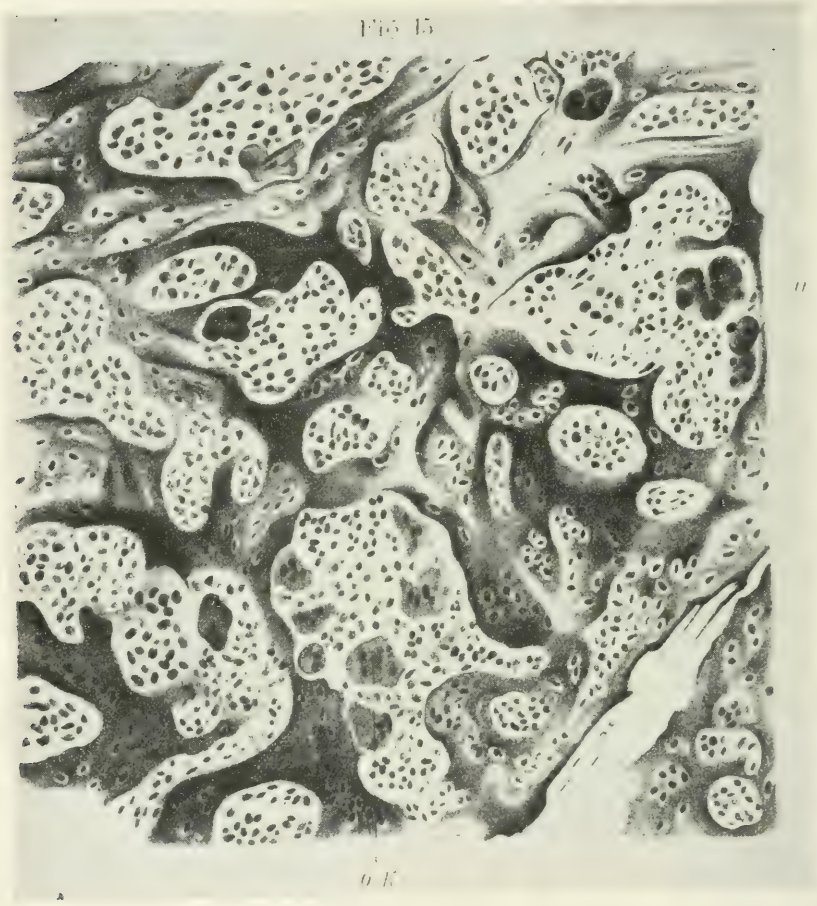


Fig. 13.

A higher magnification of the previous figure showing destruction of the new formed bone as the result of cribiform rarefaction. The bone is very porous. A cell is to be seen in each cavity. In certain places the cavities have separating walls which are very thin. In other spaces these are lacking and they are confluent, forming streets or large spaces on the border adjacent to the normal bone. M. K. No cells are to be seen. (From Manasse's collection with permission.)

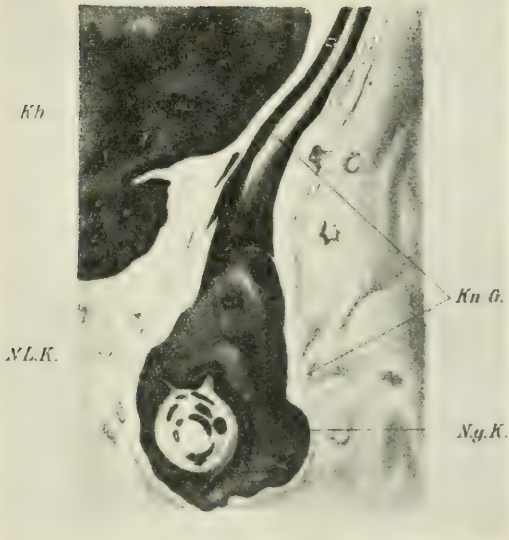
6. *Plate 11—Fig. 15 -Case 5. Right*



Section through the recent portions of large new bony growths. Extensive destruction of the dark colored new tissue partly as the result of the Howschipschen lacunae and osteoclasts (O. K.) and partly the result of cribiform rarefaction of the bone substance seen on the right hand side below. On the left again new formed bone. (From Manasse's collection with permission.)

7. *Fig. 16—Case 4. Right.*

Fig. 16.



A higher magnification. Blood vessel in the bone. (Kn. G.) Within the normal labyrinthine bone. (Nl. K. M.) The vicinity of the new formed bone. (K. h.) On the outer wall of the blood vessel we see new formed, very young bone tissue, (N. g. K.), which presses in the form of hummocks against the old bone and the blood vessel which are thus being caused to disappear. (From Manasse's collection with permission.)

OCULAR MANIFESTATIONS IN NASAL AND AURAL DISEASES WHICH PROBABLY INDICATE INVOLVEMENT OF THE SYMPATHETIC NERVOUS SYSTEM.

By WILLIAM H. HASKIN, M. D., New York, N. Y.

In recent years a great deal has been written about the ocular symptoms in both aural and nasal diseases, and below is given a brief summary of the many conditions which have been described by the various writers. It is unfortunate that there has been no definite system of differentiating the various symptoms, especially when considering the optic nerve. There is an absolute difference between optic neuritis and choked disc and care should be taken not to describe the one when the other is really present. The writer has roughly grouped the numerous symptoms given as follows:

1. Amblyopia, amaurosis, amaurotic-amblyopia, scotomas, and contracted fields.

2. Discs: Hyperaemic, outlines pale and indistinct, congestion, margins of the nerve indistinct or hazy with hyperaemia of the disc.

3. Papillae: White and atrophic without showing optic neuritis, prepapillary edema, papillitis.

4. Retina: Retinitis, retino-choroiditis, edema of, detachment of, edema with exudate, swelling and tortuosity of the retinal vessels, vessels overful, general edematous condition of the fundus, hyperaemia of the arteries, pulsation of the arteries and veins.

5. Unilateral and bilateral neuritis, retrobulbar and perineuritis. Vitreous opacities, exopthalmos, increased intraocular tension, edema of the upper lids, dilatation of the pupil, oculomotor paralysis, abducens paralysis, disturbances of lachrymation, photophobia.

In addition to these symptoms, pain is generally present, although it varies greatly in character. Dr. Sluder has also noticed disturbances of taste and has noted paralysis of the palate muscles, which are supplied by the superior max-

illary branch of the fifth nerve. Intractable sneezing, profuse rhinorrhea, a tickling sensation in the palate and areas of anaesthesia have also been described.

Dr. Wolf Freudenthal has reported a significant case of angio-neurotic edema of the face occurring in a patient upon whom he had previously operated for frontal sinusitis. Five years before operation she had suffered for a year with a profuse hydrorrhoea nasalis of the left nostril only, which had not responded to treatment but finally disappeared of itself. This was undoubtedly a case of true involvement of the sympathetic.

The relationship of the optic nerve to the post-ethmoidal cells and to the sphenoidal sinuses has been very thoroughly described by many, but very few have apparently noticed the intimate association existing between the fifth nerve and the sphenoids. Drs. Sluder and Onodi have also noted the proximity of the other orbital nerves to the sphenoid sinuses and the possibility of their involvement in disease of the sinuses. The writer has never found these relationships with the exception of the second branch of the fifth nerve, which is very frequently and extensively in contact with the sinus, both the sensory portion and the motor and the sympathetic, i. e., the Vidian portion, lying on a portion of the sinus where they would be more likely to be involved, if the sinus were inflamed than even the optic. (See illustration No. VIII.) The position of the internal carotid with its acute turns, first bending forward as it enters the cavernous then upwards and inwards at the anterior clinoid process, must keep the sinus wall away from the sphenoid and the nerves all lie in the outer wall of the cavernous. The writer wishes to add the sympathetic nerves to the numerous relations already noted and to emphasize the very intimate contact that exists between these nerves and the bony walls of the sinuses. As the majority of the symptoms (ocular and nasal) are due to vaso-motor disturbances and as the sympathetic nerves are the vaso-motor nerves, it seems to the writer that we have a most reasonable explanation for most of these manifestations

in that they are caused by irritation of this system. An examination of the specimens will show how slightly any relationship can exist between the third, the fourth, the first branch of the fifth or the sixth nerves. They all enter the cavernous sinus external to the carotid artery (which latter extends the whole length of the sinus and lies on its inner wall), being covered by its endothelial lining membrane, except the sixth, which lies on the external wall of the artery. They all enter the orbit from this outer wall passing through the common tendon of the orbital muscles and the writer has yet to see a specimen showing any actual relationship of these nerves and the sinuses. With the second branch of the fifth, however, the relationship is very intimate as said before and the third branch is frequently in relation with the sinus as can be shown on the specimens. Dr. Sluder even claims to have seen the Vidian nerve actually exposed in the sinus.

The writer has never been satisfied with the various theories in explanation of the many ocular symptoms in these cases in which there are no intracranial complications, orbital abcesses or even pressure within the diseased bone sinuses themselves. He has been unable to find any reports of microscopic proof of neuritis, except where meningitis was present. It is well recognized that intracranial pressure produces characteristic disturbances and optic neuritis, brain tumors, gummata and subdural or brain abcesses are among the many things to be remembered and to be determined in our efforts to interpret the eye symptoms. The writer desires to speak only of those cases in which the ocular disturbances are relieved by treatment of the nasal sinuses. In order to fully appreciate the reasonability of the writer's views, a complete description of the anatomy and physiology of these nerves is given below, being taken from Gray's "Anatomy" and from Landois' "Physiology."

The third nerve anastomoses are: (a) on the cavernous sinus with the first branch of the fifth receiving its sensory fibres supplying the iris; (b) with the sympathetic through the carotid plexus and (c) indirectly with the sixth, whereby

it receives its vaso-motor fibres. It contains (1) voluntary motor fibres for all the orbital muscles except the external rectus and the superior oblique. (2) The fibres of the sphincter pupillae, which are excited reflexly from the retina. (3) The fibres for the tensor choroidae which is the muscle of accommodation. The two latter sets form the short root of the ciliary ganglion and reach the eyeball through the ciliary nerves. This nerve controls the pupillary action associated with accommodation. Stimulation of this nerve causes contraction of the pupil while paralysis causes dilatation of the pupil. It should be remembered that in paralysis of the third nerve, the pupil will not contract either for light or for accommodation, whereas stimulation causes contraction.

The fourth nerve anastomoses with the carotid plexus and with the first branch of the fifth. It supplies the superior oblique muscle.

In describing the 5th nerve, it should be borne in mind that the origins of its sensory root anastomose with the motor nuclei of all the nerves arising from the medulla oblongata, except that of the sixth, as this fact explains the vast number of reflex relations of this nerve. As it crosses over the superior border of the petrous portion of the temporal bone, it is in direct contact with the superior petrosal sinus. It then becomes enlarged as the Gasserian ganglion and lies directly over the carotid canal of the petrous bone and receives sympathetic fibres from the carotid plexus. Landois says that it also receives fibres from the cavernous plexus. The following description of the 1st and 2nd branches is given from Landois and should be carefully studied. (See illustration No. II).

I. The ophthalmic division receives sympathetic fibres (vaso-motor nerves) from the plexus cavernosus; it passes through the superior orbital fissure (sphenoidal) into the orbit. Its branches are:

1. The small recurrent nerve which gives sensory branches to the tentorium cerebelli. Fibres—the vaso-motor nerves for the dura mater—proceed along with it from the carotid plexus of the sympathetic.

2. The lachrymal nerve gives off—(a) Sensory branches to the conjunctiva, the upper eyelid, the neighboring part of the skin over the temple; (b) true sensory fibres to the lachrymal gland. Stimulation of this nerve is said to cause a secretion of tears, while its section prevents the reflex secretion excited through the sensory nerves of the eye. After a time, section of the nerve is followed by a paralytic secretion of tears, although the statement is contested by Reich. The secretion of tears may be excited reflexly, by strong stimulation of the retina by light, by stimulation of the first and second branches of the trigeminus, and through all the sensory cranial nerves.

3. The frontal (f)* gives off the supratrochlear, which supplies sensory fibres to the upper eyelids, brow, glabella, and those which excite the secretion of tears reflexly; and by its supraorbital branch (b), analogous branches to the upper eyelid, skin of the forehead, and the adjoining skin over the temple as far as the vertex.

4. The naso-ciliary nerve (nc) by its infratrochlear branch supplies fibres, similar to those of 3, to the conjunctiva, caruncula, and sacculus lachrymalis, the upper eyelid, brow, and root of the nose. Its ethmoidal branch supplies the tip and alae of the nose, outside and inside, with sensory branches, as well as the upper part of the septum and the turbinated bones with sensory fibres, which can act as afferent nerves in the reflex secretion of tears; while it is probable that vaso-motor fibres are supplied to these parts through the same channel. (These fibres may be derived from the anastomosis with the sympathetic). The naso-ciliary nerve gives off the long root (l) of the ciliary (c) and 1 to 3 long ciliary nerves. (See illustration No. 1).

The ciliary ganglion, which according to Schwalbe, perhaps belongs rather to the third than to the fifth nerve, has three roots—(a) the short or oculomotorius; (b) the long, (L), from the naso-ciliary; and (c) the sympathetic (s) sometimes united with b, from the carotid plexus. The short

*All letters in brackets refer to illustration No. I.

ciliary nerves (t), six to ten in number, proceed from the ganglion, along the long ciliary nerves, to near the entrance of the optic nerve, where they perforate the sclerotic coat and run forwards between it and the choroid. Tiedemann says that it also communicates with Meckle's.

Ciliary nerves.—physiologically these nerves contain:—

1. The motor fibres of the sphincter pupillae and the tensor choroidae from the root of the oculomotorius.

2. Sensory fibres of the cornea, which are distributed as excessively fine fibrils between the epithelium of the conjunctiva bulbi; they perforate the sclera. These fibres cause a reflex secretion of tears (N. lachrymalis) and closure of the eyelids (N. facialis). Sensory fibres are supplied to the iris (pain in iritis and operations on the iris), the choroid (painful tension when the ciliary muscle is strained), and the sclera.

3. Vaso-motor nerves for the blood vessels of the iris, choroid, and retina. They arise in part from the sympathetic root and the anastomosis of the sympathetic with the ophthalmic division of the trigeminus. The iris and retina receive most of their vaso-motor nerves from the trigeminus itself, and a few from the sympathetic direct.

4. Motor fibres for the dilator pupillae, which for the most part are derived from the sympathetic, through the sympathetic root of the ganglion and the anastomosis of the sympathetic with the trigeminus. Some observers deny altogether the existence of a dilator pupillae muscle. The ophthalmic division contains independent fibres for the dilatation of the pupil which arise in the medulla oblongata and proceed directly into the ophthalmic or arise from the Gasserian ganglion.

After section of the trigeminus the pupil becomes contracted after a short period of dilatation (rabbit, frog), but this effect is not permanent. After excision of the superior cervical ganglion of the sympathetic, the power of dilatation of the pupil is not completely abolished. The narrowing of

the pupil which follows section of the trigeminus in the rabbit, and which rarely lasts more than half an hour, may be regarded as due to a reflex stimulation of the oculomotorius fibres of the sphincter, in consequence of the painful stimulation caused by section of the trigeminus.

Stimulation of the cervical sympathetic either in the neck or in its course to the eye; when the peripheral end of the cervical sympathetic is stimulated, besides causing constriction on the blood vessels on that side of the head, there is dilatation of the pupil, as well as contraction of the smooth muscular fibres (Müller's muscle) in the orbit and eyelids. The membrane of the inferior orbital fissure (spheno-maxillary fissure) in man has a layer of smooth muscle, one millimetre thick, and arranged for the most part longitudinally. Both eyelids contain smooth muscular fibres which serve to close them; in the upper lid they lie close under the conjunctiva. Tenon's capsule also contains smooth muscular fibres. The sympathetic nerve supplies all these muscles (the orbital muscle is partly supplied from the spheno-palatine ganglion). Hence stimulation of the sympathetic causes dilatation of the pupil and of the palpebral fissure, with protusion of the eyeball. This result may be caused reflexly by strong stimulation of sensory nerves. Strong stimulation of the nerves of the sexual organs is followed by similar phenomena in the eye. The dilatation of the pupil which occurs in children affected with intestinal worms, is perhaps an analogous phenomena. The pupil is dilated when the spinal cord is stimulated (at the origin of the sympathetic), as in tetanus.

Section of the cervical sympathetic, besides other effects, notably those on the blood vessels on that side of the head and face, causes narrowing of the fissure between the eyelids, the eyeball sinks in its socket.

5. It is possible that trophic fibres occur in the trigeminus and pass through the ciliary nerves to reach the eye. If the trigeminus be divided within the cranium, after six to eight days, inflammation, necrosis of the cornea, and ulti-

mately complete destruction of the eyeball takes place, constituting panophthalmia.

Section of the trigeminus paralyzes the vaso-motor nerves in the interior of the eyeball, which must undoubtedly cause a disturbance in the intraocular circulation. According to Jesner and Gruhagen the trigeminus also contains vaso-dilator fibres, whose stimulation is followed by increased flow of blood to the eye with consecutive excretion of the fibrin factors and an increase in the amount of albumin of the aqueous humor. After section of the nerve, the intraocular tension is diminished (while stimulation of the nerve is followed in increase of the intraocular pressure). This diminution of the normal tension necessarily must alter the normal relation of the filling of the blood and lymph vessels, and also the movement of the fluids, upon which the normal nutrition is largely dependent.

There are three conditions on which the above changes may depend: (1) mere loss of sensibility, which alone is not sufficient to explain the phenomena; (2) vaso-motor disturbance, which is excluded by the above facts, and also by the other consideration that, if the fifth nerve be divided and the superior cervical ganglion excised simultaneously, ophthalmia does not occur, and, in fact, excision of this sympathetic ganglion may modify the results of section of the fifth. Thus, we are forced to (3) the theory of trophic fibres, whose centre is the Gasserian ganglion.

Pathological.—In cases of anaesthesia of the trigeminus in man, and, more rarely, in severe irritation of this nerve, inflammation of the conjunctiva, ulceration and perforation of the cornea, and finally panophthalmia, have been observed. This condition has been called ophthalmia neuroparalytica.

There are other affections of the eye depending upon disease of the vaso-motor nerves, which are quite different from the foregoing, as they never lead to degenerative changes. Such as ophthalmia intermittens (due to malaria), a unilateral, intermittent, excessive filling of the blood vessels of the eye, accompanied by the secretion of tears,

photophobia, often accompanied by iritis and effusion of pus into the chambers of the eye. This condition is regarded by Eulenburg as a vaso-neurotic affection of the ocular blood vessels. Pathological observations, as well as experiments upon animals, have shown that there is an intimate physiological connection between the vascular areas of both eyes, so that affections of the vascular area of one eye are apt to induce similar disturbances of the opposite eye. This serves to explain the fact that inflammatory processes in the interior of one eyeball are apt to produce a similar condition in the other eye. This is the so-called "sympathetic ophthalmia." It probably is affected through Ribe's ganglion. Thus, stimulation of the ciliary nerves, or the fifth on one side causes dilatation of the blood vessels not only on its own side but also on the other side as well. The pathological condition of glaucoma simplex, where the intraocular tension is greatly increased, is ascribed by Donders to irritation of the trigeminus. (Increased intraocular tension may be produced by irritation of the secretory fibres contained in the fifth nerve, by stimulating the nucleus of the trigeminus in the medulla oblongate, and also reflexly by irritation of the peripheral branches of the fifth, as by nicotin placed in the eye. It is possible, however, that some forms of glaucoma are produced by diminished removal of the aqueous humor from the eye). Unilateral secretion of tears, due to irritation of the ophthalmic division of the fifth, has been repeatedly observed, but unilateral cessation of tears, due to paralytic conditions very rarely. (See illustration No. III).

II. SUPERIOR MAXILLARY DIVISION.—It gives off:

1. The delicate recurrent nerve, a sensory branch to the dura mater, which accompanies the vaso-motor nerves, derived from the superior cervical ganglion of the sympathetic, and is distributed to the area of the middle meningeal artery.

2. The subcutaneous malar or orbital (o) supplies by its temporal and orbital branches sensibility to the lateral angle

of the eye and the adjoining area of skin of the temple and cheek. Certain fibres are said to be the true secretory nerves for tears.

3. The dental, anterior, posterior, and median, and with them the anterior fibres from the infraorbital nerve, supply sensory fibres to the teeth in the upper jaw, the gum, periosteum, and the cavities of the jaw. The vaso-motor nerves of all these parts are supplied from the upper cervical ganglion of the sympathetic.

The spheno-palatine ganglion (Meckel's —n) forms connections with the second division. To it pass two short sensory root-fibres from the second division itself, which are called spheno-palatine. Motor fibres enter the ganglion from behind, through the large superficial petrosal branch of the facial (j); and the gray vaso-motor fibres (v) from the sympathetic plexus on the carotid (the deep, large petrosal nerve). The motor and vaso-motor fibres from the Vidian nerve, which reach the ganglion through the canal of the same name.

Branches of Meckel's Ganglion.—(1) The sensory fibres (N) which supply the roof, lateral walls, and septum of the nose (posterior and superior nasal); the terminal fibres of the naso-palatine pass through the canalis incisivus to the hard palate, behind the incisor teeth. The sensory inferior and posterior nasals for the lower and middle turbinated bones, and both lower and nasal ducts, are derived from the anterior palatine branch of the ganglion which descends in the palato-maxillary canal. Lastly, the sensory branches of the hard (p) and soft palate (pl) and the tonsils arise from the posterior palatine nerve. All the sensory fibres of the nose (see also the Ethmoidal nerve), when stimulated, cause the reflex act of sneezing. Preparatory to the act of sneezing, there is always a peculiar feeling of tickling in the nose, which is perhaps due to dilatation of the nasal blood-vessels. This dilatation is rapidly caused by cold, more especially when it is applied directly to the skin. The dilatation of the vessels is followed by increased secretion of watery fluid from the nasal mucous membrane. Stimula-

tion of the nasal nerves also causes a reflex secretion of tears, and it may also cause stand-still of the respiratory movements of the expiratory phase. (2) The motor branches descend in the posterior palatine nerve through the small palatine canal, and give off (h) motor branches to the elevator of the soft palate and azygos uvulae. The sensory fibres of these muscles are supplied by the trigeminus. According to Politzer, spasmodic contraction of these muscles occasionally causes crackling noises in the ears. (3) The vaso-motor nerves of this entire area arise from the sympathetic root; i. e., from the upper cervical ganglion, the root of the trigeminus supplies the secretory nerves of the mucous glands of the nasal mucous membrane. Thus, trophic functions for the mucosa have been ascribed to the trigeminus. (See illustration No. V).

The 3rd branch, with its otic ganglion, is rarely involved in the conditions we are studying, supplying as it does the lower part of the face and neck. It is closely associated, however, with the other branches through its ganglion and through the tympanic plexus, which accounts for the pains which are often reflected to the ear when the 5th nerve itself is really the site of the lesion. Below is given the anatomy of the cephalic portion of the sympathetic, being taken from Gray's anatomy.

THE SYMPATHETIC NERVE.

The Sympathetic Nerve is so called from the opinion entertained that through it is produced a sympathy between the affections of distant organs. It consists of a series of ganglia, connected together by intervening cords, extending on each side of the vertebral column from the base of the skull to the coccyx. It may, moreover, be traced up into the head, where the ganglia (which are all in connection with the fifth cranial nerve) occupy spaces between the facial and the cranial bones.

The sympathetic nerve, for convenience of description, may be divided into several parts, according to the position

occupied by each. Of these, we will briefly describe:—

1. Cephalic portion.....4 pairs of ganglia.
2. Cervical portion.....3 pairs of ganglia.

Each ganglion may be regarded as a distinct centre, from or to which branches pass in various directions. These branches may be thus arranged: 1. Branches of communication between the ganglia. 2. Branches of communication with the cerebral or spinal nerves. 3. Primary branches passing to be distributed to the arteries in the vicinity of the ganglia, and to the viscera, or proceeding to other ganglia placed in the thorax, abdomen or pelvis.

Of these, the primary branches of distribution also consist of two kinds of nerve-fibres: the sympathetic and the spinal. They have a remarkable tendency to form intricate plexuses which encircle the blood-vessels, and are conducted by them to the viscera.

The *cephalic portion* of the sympathetic consists of four pairs of ganglia: 1. The ophthalmic ganglion. 2. The spheno-palatine, or Meckel's ganglion. 3. The otic, or Arnold's ganglion. 4. The sub-maxillary ganglion. These have been already described in connection with the three divisions of the fifth nerve. There is also the single ganglion uniting the two sides; i. e., Ribe's ganglion.

CERVICAL PORTION OF THE SYMPATHETIC.

The cervical portion of the sympathetic consists of three ganglia on each side, which are distinguished, according to their position, as the superior, middle, and inferior cervical.

The *superior cervical ganglion**, the largest of the three and the one which is of interest to us, is placed opposite the second and third cervical vertebrae and sometimes as low as the fourth or fifth. It is of a reddish-grey color, and usually fusiform in shape; sometimes broad and occasionally constricted at intervals, so as to give rise to the opinion that it consists of the coalescence of several smaller ganglia. It is in relation, in front, with the sheath of the internal carotid

*See illustration No. 1.

artery and internal jugular vein; behind, it lies on the Rectus capitus anticus major muscle.

Its branches may be divided into superior, inferior, external, internal and anterior.

The *superior branch* appears to be a direct continuation of the ganglion. It is soft in texture and of a reddish color. It ascends by the side of the internal carotid artery and, entering the carotid canal in the temporal bone, divides into two branches, which lie, one on the outer, and the other on the inner side of that vessel.

The *outer branch*, the larger of the two, distributes filaments to the internal carotid artery, and forms the carotid plexus.

The *inner branch* also distributes filaments to the internal carotid, and, continuing onwards, forms the cavernous plexus. (See illustration No. VI).

Carotid Plexus.

The carotid plexus is situated on the outer side of the internal carotid. Filaments from this plexus occasionally form a small gangliform swelling on the under surface of the artery, which is called the carotid ganglion. The carotid plexus communicates with the Gasserian ganglion, with the sixth nerve, and spheno-palatine ganglion, and distributes filaments to the walls of the carotid artery, and to the dura mater, (Valentin, see illustration No. VII), while in the carotid canal it communicates with Jacobson's nerve, the tympanic branch of the glossopharyngeal.

The *communicating branches* with the sixth nerve consist of one or two filaments which join that nerve as it lies upon the outer side of the internal carotid. Other filaments are also connected with the Gasserian ganglion. The communication with the spheno-palatine ganglion is effected by the carotid portion of the Vidian nerve, which passes forwards, through the cartilaginous substance filling the foramen lacerum medium, along the pterygoid or Vidian canal to the spheno-palatine ganglion. In this canal it joins the petrosal branch of the facial, thus forming the Vidian.

Cavernous Plexus.

The cavernous plexus is situated below, and internal to that part of the internal carotid which is placed by the side of the sella Turcica, in the cavernous sinus, and is formed chiefly by the internal division of the ascending branch from the superior cervical ganglion. It communicates with the third, fourth, fifth and sixth nerves, and with the ophthalmic ganglion, and distributes filaments to the wall of the internal carotid. The branch of communication with the third nerve joins it at its point of division; the branch to the fourth nerve joins it as it lies on the outer wall of the cavernous sinus; other filaments are connected with the under surface of the trunk of the ophthalmic nerve; and a second filament of communication joins the sixth nerve.

The filament of connection with the ophthalmic ganglion arises from the anterior part of the cavernous plexus; it accompanies the nasal nerve, or continues forwards as a separate branch.

The terminal filaments from the carotid and cavernous plexuses are prolonged along the internal carotid, forming plexuses which entwine round the cerebral and ophthalmic arteries; along the former vessel they may be traced on to the pia mater; along the latter, into the orbit, where they accompany each of the subdivisions of the vessel, a separate plexus passing from the arteria centralis retinae into the interior of the eyeball. The filaments prolonged on to the anterior communicating artery form a small ganglion, *the ganglion of Ribes*, which serves, as mentioned above, to connect the sympathetic nerves of the right and left sides.

If we now take up the physiology of the sympathetic, it at once becomes interesting in connection with our subject. The functions attributed to the cervical portion are as follows:—

1:—Pupil dilating fibres. Section of the cervical sympathetic causes contraction while irritation causes dilatation of the pupil.

2:—Motor fibres for Müller's smooth muscle of the orbit.

3:—Vaso-motor fibres for the outer ear and side of the

face, the tympanum, conjunctiva, iris, choroid, and the retina in part.

4:—Trophic fibres, sweat fibres for the face, and secretory fibres for the various glands, such as the lachrymal, the several salivary, and the mucous glands.

With this sketch before us, the writer desires to read some extracts from a reprint recently sent to him by Dr. Gruening after having discussed the question with him. In it he says: That at times the statement is made that an optic neuritis has developed into a choked disc, thus implying that choked disc is a more advanced stage of optic neuritis." This he says is an erroneous view. He describes choked disc as having an initial, an intermediate and advanced stage, every one of which is the ophthmoscopically visible expression of increased intracranial pressure. He then tells us why it is that there is never any choked disc when an optic neuritis is present, because the intervaginal spaces are sealed by exudative material so that the serum does not enter lymph spaces of the optic nerve and the perilymphatic sheaths of the optic and retinal vessels. He then asks this pertinent question: "How can the choking of the disc, which is so far from the area of the disease, in purulent middle ear disease be explained?" He acknowledges in answer to this question that there is no succinct and lucid explanation of this inter-relation. He then asks: "But what causes the excess of the cerebrospinal fluid?" and in answer says that the theory of chemo-taxis, as defined by Theodore Leber, may furnish the clue. That theory is a most difficult one to understand and to the writer cannot explain why one orbit is so often affected and not both. Dr. Gruening also writes that another point of interest is that the degree of swelling may vary in the two discs. As we assume that the hydrostatic pressure is equal at all points within the brain cavity, there must be structural conditions causing these variations. In conclusion he says: "that further confirmation that we have not to deal with inflammatory action and destruction of the nerve tissue in choked discs in otitic cases is supplied by the fact that, on removal of the purulent area, the optic

nerve returns to the normal." When such an authority as Dr. Gruening offers such a paper before the International Otological Congress it seems to be pretty conclusive evidence that, as yet, but little is known of the true conditions which are manifested in the eyes and affords the writer some excuse for the length of the present paper. He asks that you bear well in mind:—

1:—That the sympathetic nerve is the vaso-motor nerve of the eye, that is, it controls the blood supply.

2:—That these nerves, after leaving the superior cervical ganglion, form the carotid plexus, and then on entering the cavernous sinus form the cavernous plexus and lie on its inner wall in contact with the sphenoids.

3:—That the branches after leaving these plexuses often run through small bony canals in the walls of cavities or sinuses that become inflamed, such as the Vidian, the greater and lesser petrosal nerves and the branches entering the tympanum.

Remembering the above relations and considering that almost all of the ocular disturbances are true disturbances of the vaso-motor system, it seems to the writer that the rational explanation of these symptoms lies in correctly attributing them to the sympathetic nerve. The delicate fibres of this system are much more exposed to irritation from inflammations in the accessory sinuses, and even in the ear, than any of the other nerves, and reference to the physiology will show that any or all of these ocular symptoms have actually been produced by experiments on these nerves. Dr. J. D. Richards describes two cases of pupil dilatation following extensive exenteration of the petrous bone with exposure of the carotid artery in its canal and attributed the dilatation to irritation of the plexus. In both cases, although the dilatation was very marked, still there was reaction to light and to accommodation, showing that the disturbance came through the fifth nerve. Although all writers maintain that choked disc is always caused by increased intracranial pressure, in most of our cases there

are absolutely no other symptoms of this condition present, and it is certainly remarkable that in most cases the eye conditions clear up as soon as the local nasal or aural disease is cured or the irritation removed. Again, it is a very significant fact that in many cases where operation has cured the nasal disease and the eye symptoms have been relieved, that an acute exacerbation will often be accompanied by a return of the eye symptoms, which will again disappear with the cure of the attack. True paralyses of the orbital muscles are not common and are difficult to explain.

However, the intimate relation of the sixth nerve with the inferior petrosal sinus as it pierces the dura, and then with the interior of the cavernous sinus itself accounts for its implication whenever these sinuses are thrombosed.

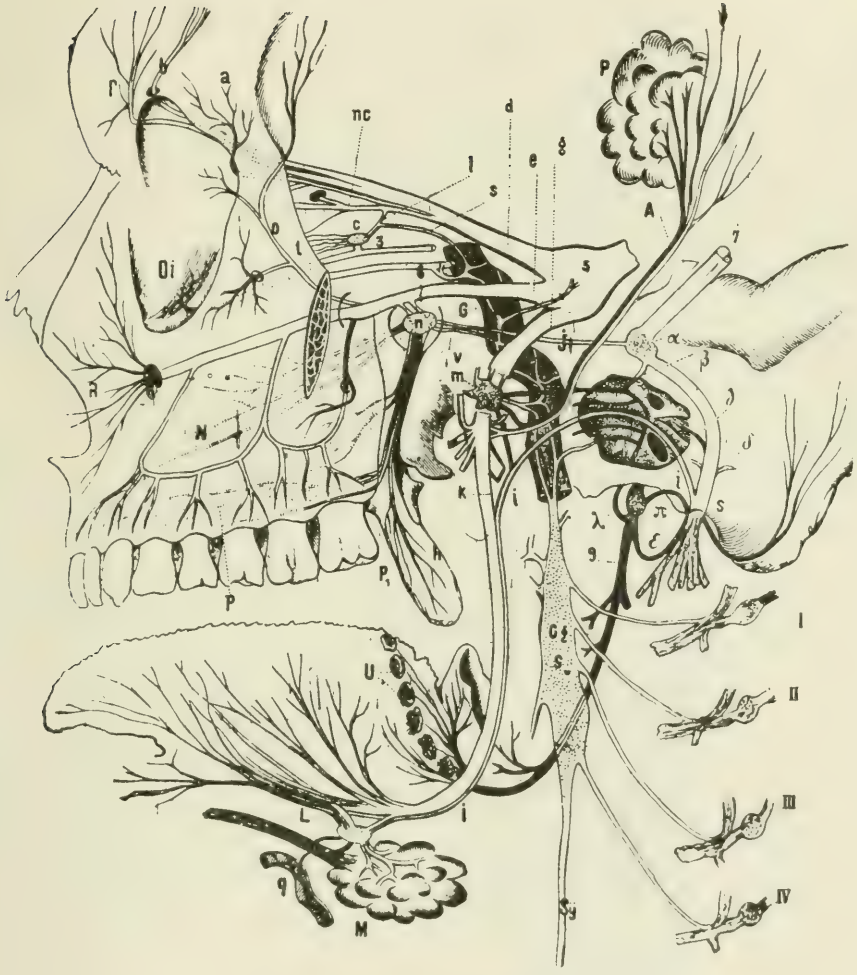
Dr. Sluder has written often concerning the spheno-palatine ganglion and his method of treating it. He has undoubtedly been very successful, but an examination of the specimens presented will show how difficult it is to reach this ganglion itself, and only an unusually skilful man with an absolute appreciation of the anatomy had better attempt these injections. It has required much time and great care for the writer to open the specimens in order to actually show the ganglion in its place where it is enmeshed in many blood-vessels.

Let us look at the disease called glaucoma. This disease consists of a great increase in the intraocular tension (due to vaso-motor disturbances) and frequently destroys the eye if not promptly relieved. Dr. Sluder reports ten cases of glaucoma in which he has relieved the pain by cocainization of the spheno-palatine foramen. Lapersonne and Fisch have reported cases of increased intraocular pressure caused by accessory sinus disease. Now turn back to the section on physiology and, under the ophthalmic branch of the fifth nerve, you will find that Donders has ascribed the greatly increased intraocular tension to irritation of the trigeminus. Read also in the same paragraph what Eulenberg says about ophthalmia intermittens, which he describes as a vaso-neurotic affection of the ocular blood-vessels. This paragraph, in con-

clusion, says that there are other diseases of the eye depending upon disease of the vaso-motor nerves which are peculiar in that they do not lead to degenerative changes. Amaurosis, i. e., weakness of the vision, and amblyopia frequently result from optic neuritis but, Landois says, that both may be the signs of disturbances in other organs, that they may be sympathetic signs due to changes in the movement of the blood-stream, depending upon stimulation of the vaso-motor nerves. In fact, a careful study of the physiological experiments upon the orbital nerves and especially the sympathetic branches will disclose the fact that all of the ocular manifestations that we see and that have been so frequently described have been caused by section of or by irritation of the sympathetic nerve supply. There is one more point the writer, in closing, wishes to suggest and that is the probable influence of the trophic nerve fibres. These are also sympathetic fibres conveying impulses from the centres to the periphery. Their end organs lie in the tissues themselves although little is known about them. They are called trophic because they are supposed to govern or control the normal metabolism of the tissues. There are many diseases attributed to disease of these fibres or the centres from which they spring, such as the various forms of herpes, the condition known as "glossy skin," progressive muscular atrophy, myxodaema (seen in the nose as polypi), leucoderma, Addison's disease, anæsthetic leprosy and acute decubitus or bed-sores. All nerves are supplied with trophic fibres and it naturally follows that if these nerves become diseased and are destroyed that the nerve supplied by them will also atrophy and lose its function. To the writer, this supplies a reasonable theory to explain why in cases of long standing disease of the sinuses, the disturbances of vision do not recover after the sinus disease has been cured, although in recent cases vision almost invariably does return. Irritation of any tissue for a short period of time will not produce any actual tissue change, but long continued irritation will gradually produce actual destruction and that is what probably does take place in these cases. At first the sympathetic nerves are

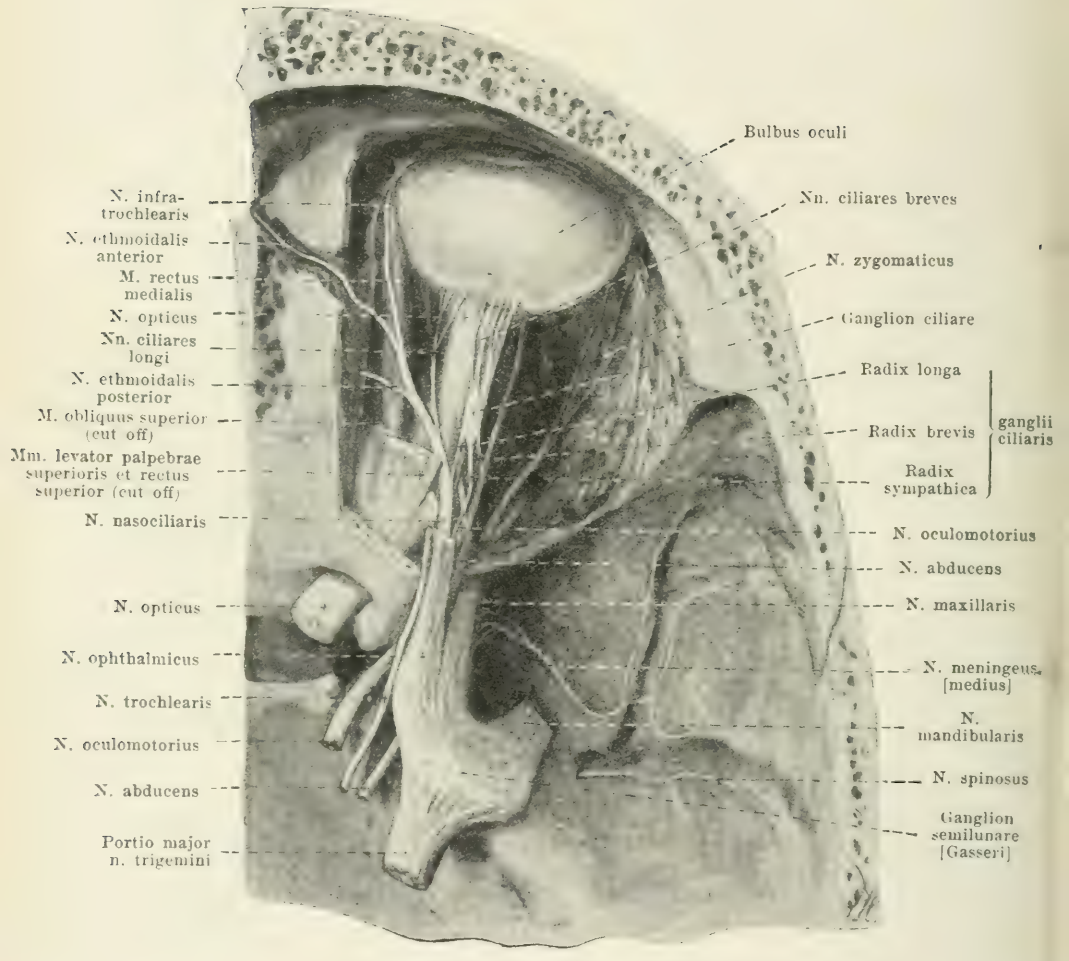
merely irritated by the inflammatory sinus and produce the symptoms of increased vaso-motor action, but after long irritation the trophic nerves become destroyed and thus destroy the trophic control of the optic and it loses its function, although an actual neuritis has never been present. When we consider that the majority of nasal affections are affections of the vaso-motor nerves and then realize that these same vaso-motor branches are so intimately associated with the sphenoid sinuses, even before they join their individual ganglia, it does not seem to be unreasonable to advance the theories advanced in this paper and the writer hopes that it will help all in the true interpretation of cases to be seen.

The writer desires to thank J. B. Lippincott & Co., and P. Blakiston's Son & Co. for their very kind permission to use the illustrations appearing in this article, which are so much better than he could have prepared himself.



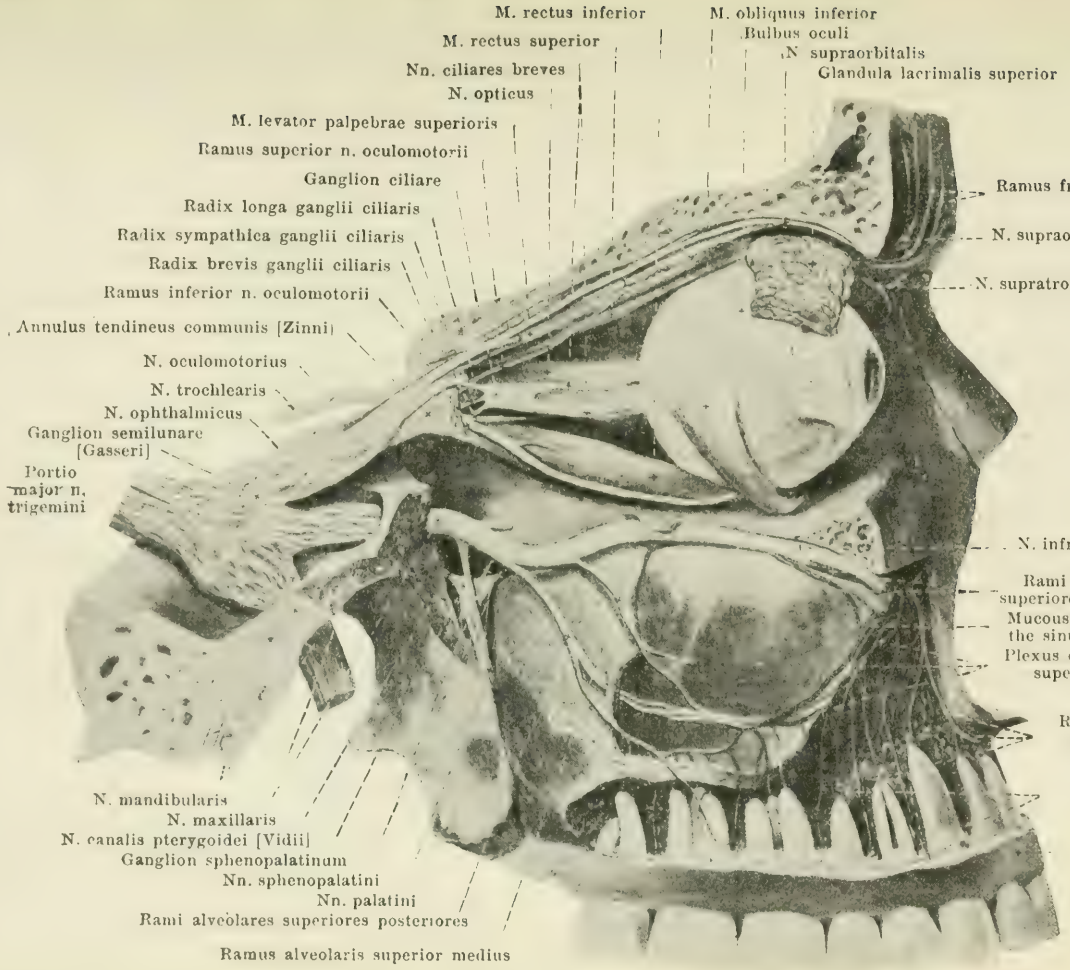
(LANDOIS PHYSIOLOGY.)

Fig. 1. Cranial sympathetic nerve anastomoses.



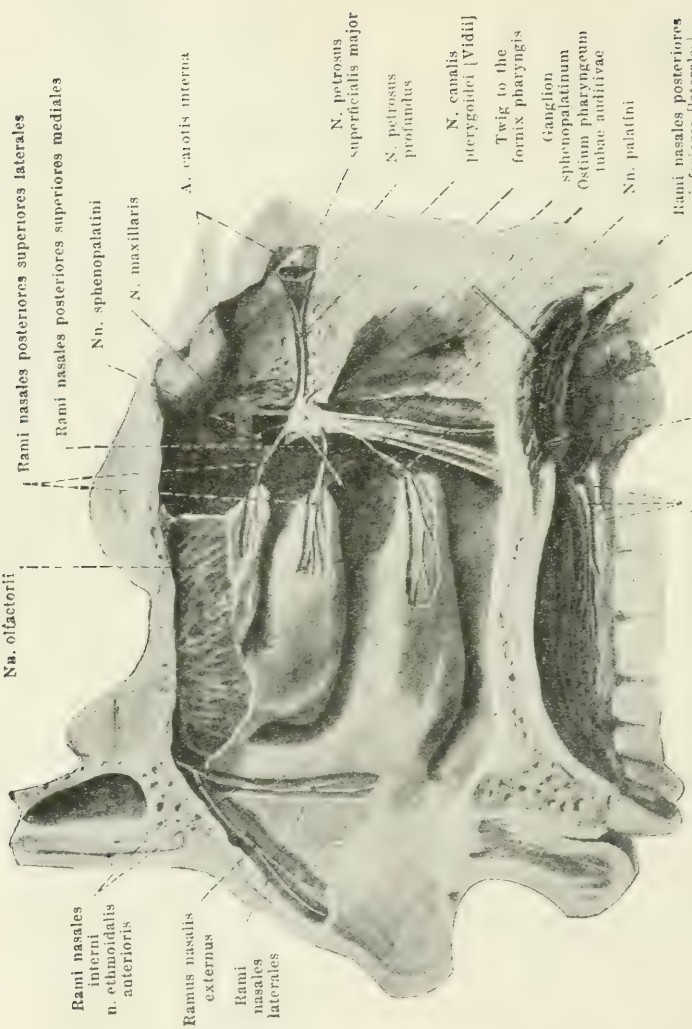
(SPALTEHOLTZ ANATOMY.)

Fig. 11. Ophthalmic branch of the fifth nerve with Gasserian and ciliary ganglia.



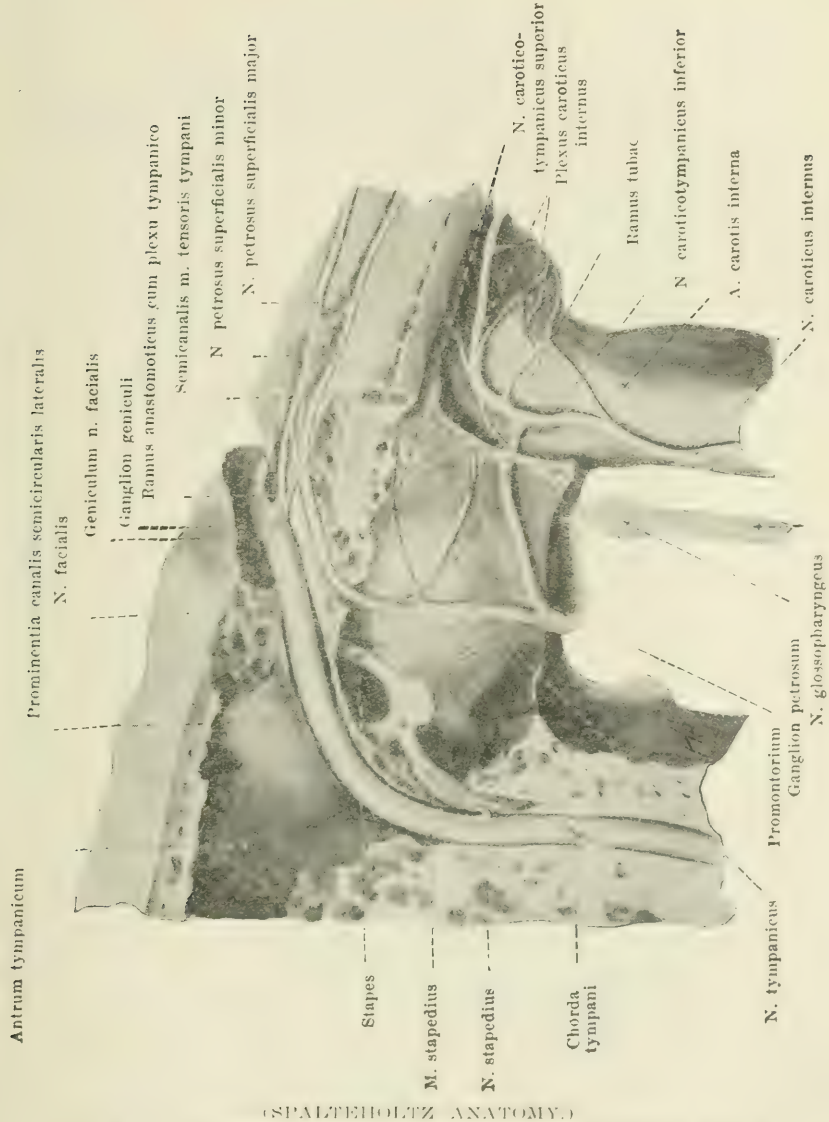
(SPALTEHOLTZ ANATOMY.)

Fig. 111. Ophthalmic and superior maxillary branches of the fifth nerve showing Gasserian, ciliary, and sphenopalatine ganglia.



(SPALTEHOLTZ ANATOMY.)

Fig. IV. Sphenopalatine ganglion with afferent and efferent branches.

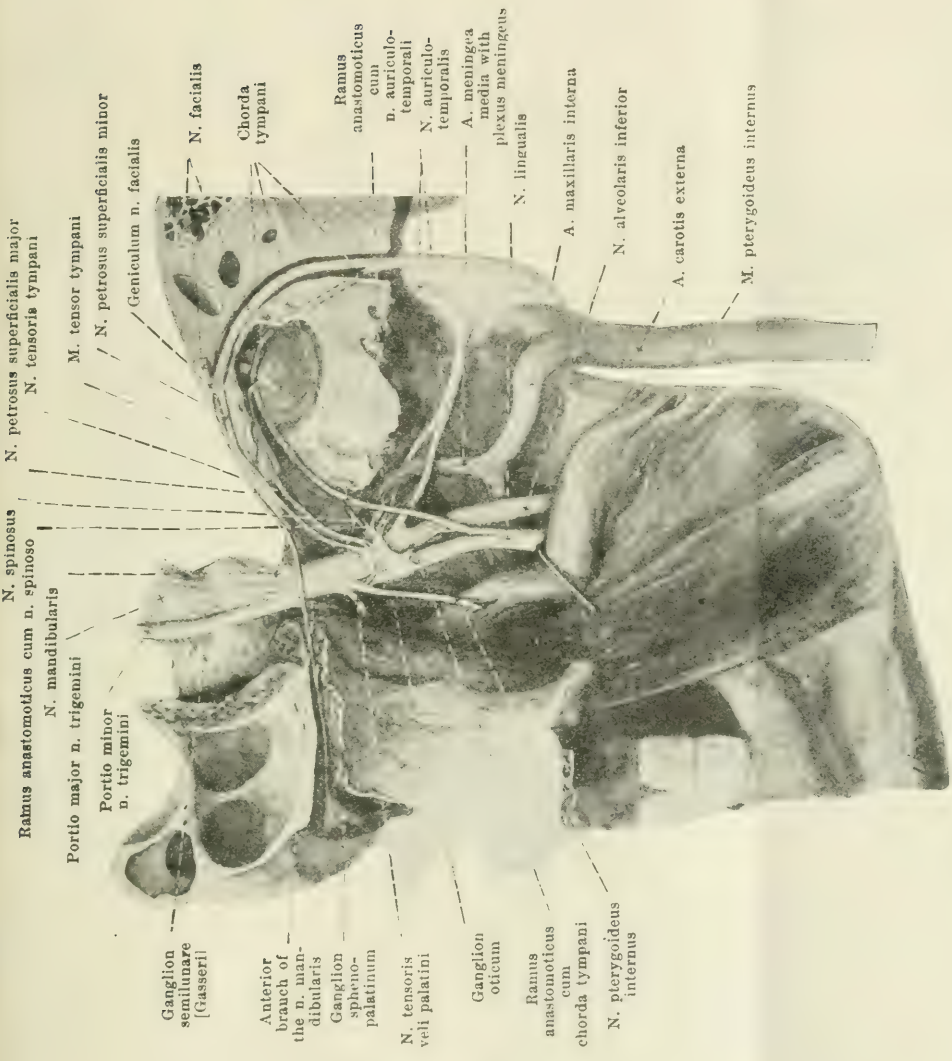


(SPALTEHOLTZ ANATOMY.)

Fig. V. Tympanic plexus anastomoses with carotid plexus geniculate and petrosal ganglia.

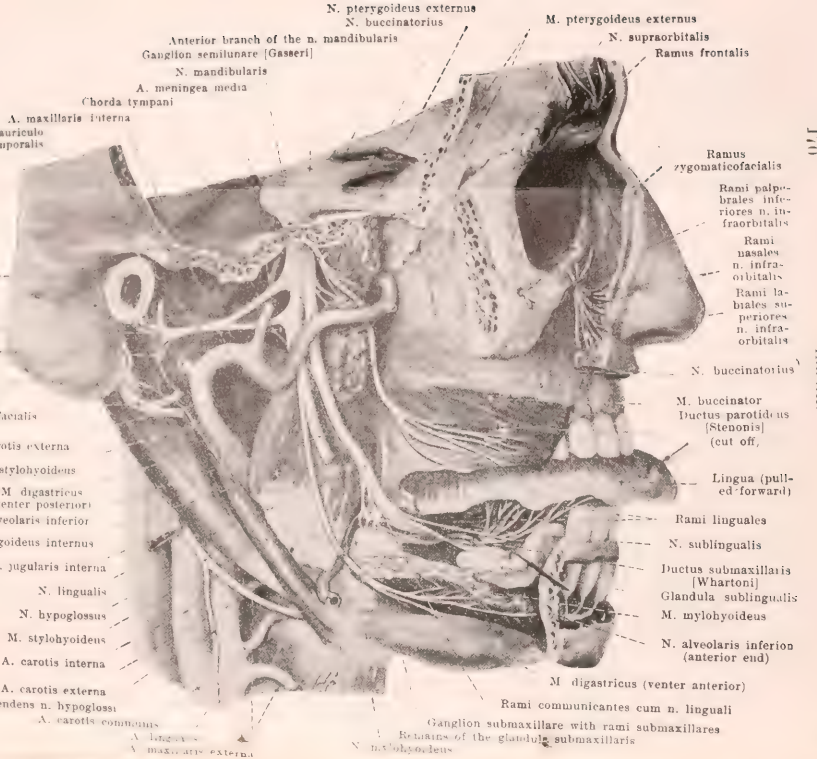
(SPALTEHOLTZ ANATOMY.)

Fig. VI. Course of the internal carotid artery with carotid and cavernous plexuses, Vidian nerve and sphenopalatine ganglion.



(SPALTEHOLZ ANATOMY.)

Fig. VII. Inferior maxillary branch of the fifth nerve showing the ganglion with its tympanic anastomoses.



Gray's Anatomy, 20th edition, 1918, Henry Gray, F.R.S., Lea & Febiger, Philadelphia

Discussion.

Dr. Max A. Goldstein, of St. Louis, Mo., congratulated Dr. Harris and Dr. Haskin for the excellent presentation of the subjects, which are certainly of intense moment in the evolution of otological work. While neither the otosclerosis problem nor the nerve question had brought out anything new and original, their discussion served to crystallize the information and to put it into form for study. There was no more difficult and complicated study in otology than that of otosclerosis. The cases presented by Siebenmann, Gradenigro, and others, at the Otological Congress, demonstrates that there is a difference between clinical otosclerosis and that which is found under the microscope. There are numerous conditions in which there may be a fixed footplate of the stapes from patholigcal changes, but which are not necessarily otosclerotic in character. These will continue to be confounded with typical otosclerosis until some better way of differentiating them is discovered. The several theories presented by Siebenmann, Dencke, and others, differ only in pathology. It seems to be definitely determined that there is a new bone formation, whatever the etiological factors producing this peculiar osteoblastic formation may be. In most of the work reported from abroad there has been found an arrest of the cartilage. From the membranic cartilage cell there is produced, by some form of stimulation, a new growth. The fact that this stimulation causes new growth is a verification of the Manasse theory. Otherwise otosclerosis would hardly go on more or less spasmodically. There are otosclerotic cases in which the process stops and then, under some stimulation, as childbirth or acute febrile conditions, there will start up a new process. This is an additional point to support the negative,—that Manasse's theory of inflammation pure and simple may be erroneous. It is strange that with the enormous amount of work done on this side of the Atlantic there is not one case studied under the microscope. He feels certains that stimulation of interest in the subject will be brought about by these papers.

Dr. Edward B. Dench, of New York City, emphasized the point that clinically it is almost impossible to make a diagnosis of otosclerosis. He believed it to be a comparatively rare condition. No man could determine, from one or two examinations, that otosclerosis is present, and consequently treatment would do no good. When impairment of hearing was moderate, whether from otosclerosis or what not, an absolutely unfavorable prognosis should not be given. There was always a possibility of improvement under local treatment.

Dr. Geo. E. Shambaugh, of Chicago, did not think it difficult to diagnose the typical cases of otosclerosis; that is, cases with bony fixation of the stapes. He pointed out that every case diagnosed in this way that had come to autopsy, showed the condition of spongyfying of the bony capsule of the labyrinth. The diagnosis is not so easy in cases where the fixation of the stapes develops in an ear which has been the seat of a long-standing otitis media, or when the otosclerosis develops without causing fixation of the stapes. In the latter case, the symptoms are those of nerve deafness. In these cases the diagnosis of otosclerosis can be surmised from the history of other cases of otosclerosis in the family, from the gradual onset of the trouble, beginning in early middle life. In this connection it is well to remember that any case of profound deafness due to obstruction in the middle ear is likely to develop a secondary degeneration in the cochlea.

WHEN TO OPERATE ON THE LABYRINTH IN LABYRINTH INFECTION SECONDARY TO PURULENT OTITIS MEDIA.

By G. E. SHAMBAUGH, Chicago, Ill.

The most difficult question in the treatment of labyrinthitis secondary to purulent otitis media, hinges on the indications for an operation on the labyrinth. A labyrinth operation in cases of labyrinth infection is called for chiefly because of the danger of a secondary intracranial complication. Invasion of the labyrinth in suppurative otitis media presents a varying clinical picture. The process may be restricted to but a part of the internal ear or it may spread throughout the entire labyrinth. The degree of inflammatory reaction in the labyrinth varies from a slight congestion, with slight alteration in the labyrinth fluids and a partial suppression of labyrinth function, to a diffuse purulent condition producing a total destruction of function. The milder cases of acute labyrinthitis, those which stop short of a complete suppression of labyrinth function, are classified usually as cases of serous labyrinthitis, while the severe cases, those which produce a complete suppression of function, are for the most part cases of empyema of the labyrinth. In the milder cases, those which produce but a partial suppression of labyrinth function, we expect, as a rule a more or less complete restoration of function after the subsidence of the labyrinthitis. In the cases of diffuse empyema of the labyrinth, there appears to be always a permanent total loss of function because the delicate sense epithelium has been entirely destroyed. Cases of acute labyrinthitis do occur, however, where there appears to be a complete suppression of function both for the cochlea and the semi-circular canals and where on the subsidence of the acute process there is at least a partial restoration of function.

An operation on the labyrinth is not indicated in the milder cases of labyrinthitis even though, as Ruttin has shown such cases may occasionally be followed by a fatal intracranial complication. It is only in cases of diffuse empyema where the function of the labyrinth has already been destroyed, that the dangers of an intracranial complication constitute an indication for a labyrinth operation.

The conditions under which an empyema of the labyrinth presents itself varies. If the process is observed soon after an acute onset it will be "manifest" by distinct active symptoms —disturbance of equilibrium and nystagmus. If it is seen first after it has existed for some time, all active symptoms may have disappeared and the process may be distinctly "latent." The labyrinth suppuration may be a sequel of an acute or a chronic purulent otitis media. If the labyrinth suppuration develops in connection with an acute otitis media it may occur as a part of a violent pan-otitis such as occasionally follows scarlet fever or measles, or it may occur secondary to the more sub-acute stage of otitis media as the sequel of a neglected mastoiditis. On the other hand, an empyema of the labyrinth may be secondary to an acute otitis media, when neither the general symptoms nor the local conditions about the ear suggest a process which would require a mastoid operation.

When an empyema of the labyrinth develops in chronic suppuration of the middle ear, it may occur in cases where the disease process has invaded the temporal bone, with or without cholesteatoma formation, that is in cases where a radical mastoid operation is already indicated; or it may develop in cases where there exists no clinical evidence of a process which calls for a mastoid operation. A diffuse suppuration of the labyrinth, moreover, may exist as a simple empyema or it may be complicated by erosion of the capsule of the labyrinth, by fistula formation into the labyrinth, by facial paralysis or by sequestration of a part or the whole of the bony capsule of the labyrinth.

The danger of an intracranial complication from an empyema of the labyrinth developing under these varying

conditions is not always the same. For example, the danger from a case of labyrinthitis which occurs as a part of a violent acute pan-otitis or from a labyrinthitis secondary to a neglected acute mastoiditis, is very much greater than when the invasion of the labyrinth occurs in a milder otitis media without distinct evidence of a mastoid involvement. In the same way an empyema of the labyrinth occurring in cases of chronic otitis media where there already exists an invasion of the temporal bone or a cholesteatoma seldom heals spontaneously and is therefore much more likely to lead sooner or later to an intracranial complication than when it develops in a case of chronic otitis media where the clinical indications for a radical mastoid operation are absent. An empyema of the labyrinth, complicated by erosion of the labyrinth capsule and fistula formation, facial paralysis or sequestration, seldom heals spontaneously and is much more dangerous than a simple uncomplicated empyema.

In view of these clinical facts it is apparent that the indication for a labyrinth operation is much more urgent in certain cases of empyema of the labyrinth than in others. The situation is quite similar to the question of the indications for the radical mastoid operation in the treatment of chronic purulent otitis media. In both conditions grave danger exists of serious intracranial complications. There was a time when this danger in chronic purulent otitis media seemed at least to some to constitute an indication in most of these cases for the radical mastoid operation. Otology has now advanced from this position to the place where we are able to make a more or less sharp distinction between those cases of chronic purulent otitis media where the danger from an intracranial complication is such as to constitute an indication for the radical mastoid operation and the cases where this operation need not be done. In the same way there are those today who would advocate the labyrinth operation for all acute cases of labyrinth infection severe enough to cause a total suppression of labyrinth function. The question arises whether we are able to make a distinction between those cases of labyrinth

empyema where the danger of an intracranial complication is sufficient to constitute an indication for the labyrinth operation and other cases of labyrinth empyema where this operation need not be done. Such a question can be settled only by the accumulated experience from the study of a large number of cases of labyrinth empyema. It is well to remember that accurate observation of cases of labyrinth infection is still comparatively new and that we have as yet hardly reached the stage where definite indications can be laid down to cover all cases. In view of the large number of cases of labyrinth empyema which recover spontaneously it is extremely desirable to make a distinction between those cases where a spontaneous recovery is extremely unlikely, that is those in which the danger of a complication is grave, and the cases where the labyrinth operation may at least be delayed with relative safety awaiting the first evidence of a further extension.

As pointed out above, the labyrinth operation is not called for in cases of labyrinthitis secondary to purulent otitis media, when the function of the internal ear has not been completely destroyed. In this connection, it should be remembered that a circumscribed suppuration of the labyrinth may occur which destroys the vestibular apparatus without involving the cochlea and which may lead to a fatal intracranial complication without at any time destroying the hearing. Such cases should be treated conservatively unless evidence of an intracranial extension appears when the labyrinth operation must be resorted to even though the operation will destroy the remnant of hearing in that ear. The same method of procedure should be applied to all the cases of labyrinthitis which completely destroy the function of the internal ear, whether they occur secondary to an acute otitis media or in connection with an acute exacerbation of a chronic otitis media, provided no clearly recognized indications exist for a mastoid operation. Furthermore a labyrinthitis which develops after a mastoid operation even when it results in complete suppression of function, should be

treated conservatively unless symptoms develop indicating an intracranial extension.

On the other hand the cases of labyrinthitis where a labyrinth operation seems to be clearly called for, include:

First, cases of labyrinth suppuration where clinical symptoms exist, suggesting a beginning intracranial complication, such as altered cerebrospinal fluid, severe unilateral headache, etc.

Second, cases where the labyrinth empyema develops as a part of a violent acute pan-otitis where the indications for a mastoid operation exist.

Third, cases where the labyrinth suppuration develops as a sequel to chronic purulent otitis media, where well recognized indications for a radical mastoid operation exist.

Fourth, cases where the labyrinth suppuration is complicated by erosion of the labyrinth capsule by fistula formation into the labyrinth, by facial paralysis or by sequestration of a part or the whole of the labyrinth capsule.

Discussion.

Dr. Edward B. Dench, of New York City, considered Dr. Shambaugh's paper a very important one. As he had said, in discussing Dr. McKimmie's paper, otologists a few years ago were all too anxious to open the labyrinth. This was not because they were over-zealous to open the labyrinth, but because they did not have enough accumulated evidence to be able to judge which cases might clear up and which might not. He did not feel that, in a given number of cases, one could tell which would clear up and which would not. If one waited until there was evidence of beginning meningeal involvement failure was almost certain, because meningitis secondary to inspection of the labyrinth is one of the most fatal diseases with which one has to deal. He had found that where there is beginning involvement there is a slight rise in temperature, but this will fall in twenty-four to forty-eight hours if nature is to throw off the infection. If the temperature remained high operation was

indicated. The temperature was a valuable guide in cases of this kind; in addition to this, the condition of the labyrinth as shown by the caloric and other functional tests.

Dr. Wendell C. Phillips, of New York City, was interested to see the extreme caution exercised by Dr. Shambaugh, also that he was still undecided as to the indications. Some of these questions were not yet settled. He was more and more inclined not to operate unless the symptoms are severe. He had had one or two unfortunate experiences in doing the radical mastoid operation in cases in which there were labyrinthine symptoms. He wished to add a word of caution with reference to doing the radical mastoid operation in the presence of acute purulent labyrinthitis. The unpleasant experiences to which he referred, he believed, were due to chiseling unnecessarily in the radical mastoid operation. Careful records should be kept of all labyrinth cases, whether operated upon or not. He and his assistants had reported twenty cases, drawing no conclusions, but simply giving the histories as they were, with the symptoms, treatment, etc. If a series of two or three hundred cases could be obtained conclusions might be drawn from them. He was more and more convinced that one should operate rather late, even though he might be criticized for delaying operation too long. In cases in which he had had the best results he had not operated at all.

Dr. Shambaugh in closing discussion, said that the position he has been lead to take could be very well illustrated by the following three cases:

The first was a case of acute otitis media in a young man. Dr. Shambaugh saw the patient about two weeks after the onset, when the patient was referred to him because of symptoms of acute labyrinth disturbance. The examination failed to disclose any distinct evidences of a mastoiditis. There were, however, all the symptoms of a complete unilaterial destruction of the labyrinth but nothing suggesting an intracranial extension. No operation was performed. The acute

middle ear process subsided in a few weeks and when seen a year after showed a completely healed middle ear, but a dead labyrinth.

The second case was one of acute labyrinthitis in a woman thirty or thirty-five years of age, who gave a history of recurring attacks of discharge from one ear, dating from childhood. Dr. Shambaugh was called to see the case one week after the acute onset, because of the extreme vertigo and nausea. The examination disclosed a complete suppression of function in one labyrinth. There was a large central perforation and the discharge chiefly mucus with no odor. There was nothing to suggest an intracranial extension and no operation was performed. The patient made the usual quick recovery from the acute otitis media, but there was no return of function in the labyrinth.

The third case was that of a young women suffering from a cholesteatoma in the middle ear, for which a radical mastoid operation was performed. Everything seemed to be doing well until the 5th day after the operation, when there developed an acute labyrinthitis, associated with a complete suppression of function in this ear. The case was watched carefully, but no symptoms developed suggesting an intracranial extension. The case made a quick recovery from the radical mastoid, but the labyrinth remained permanently dead.

It would seem that all three of these cases were probably cases of empyema of the labyrinth. The question of operating upon the labyrinth for a labyrinth empyema is quite different if the case is one where a mastoid operation is clearly indicated. Here it would seem that the mastoid operation should be followed by a thorough exentration of the labyrinth.

A REPORT OF THREE CASES OF OTITIC MENINGITIS TREATED BY DRAINAGE OF THE CISTERNA MAGNA.

By EDWARD BRADFORD DENCH, M. D., New York, N. Y.

The few remarks which I have to make can hardly be dignified by the title of "a paper." They are simply a report of a personal experience with an operative procedure, proposed just about one year ago, and for which much was hoped. I refer to the drainage of the cisterna magna for the relief of meningitis. The operation is not a new one, as a procedure for draining this region is mentioned by Cunningham (Textbook of Anatomy, 1902, p. 1164). This author not only describes a means of draining the cisterna, but also of opening the fourth ventricle. To Haynes, however, (Transactions of the American Laryngological, Rhinological and Otological Society, 1912), is undoubtedly due the credit of perfecting the technique of this operation, and of bringing it before the profession as a definite procedure for the relief of meningitis.

I simply wish to speak of three cases upon which this operation was performed. It is not necessary to give a detailed history of these cases. I will, therefore, only burden you with a brief resumé of the history in each case:

Case I. A child three and a half years of age. The history was briefly that the child had been out of sorts for about a month. A week before I saw the child, there was spontaneous discharge from the left ear. Three days later there was a rise in temperature. For the twenty-four hours immediately preceding my visit, the child had complained of pain in the head and in the left ear, and had cried out at irregular intervals. There was some discharge from the left external auditory canal. An examination of the ear showed a large perforation, involving the entire lower portion of the drum membrane, the mucous membrane of the tympanic cavity being red, but not polypoid in character.

A bacteriological examination of the discharge showed streptococci. No tubercle bacilli were found in the discharge. There was no tenderness over either mastoid. The right ear was normal. An examination of the child showed slight rigidity of the entire body, but this was not marked. No Babinski sign. Kernig sign present on right side, but not marked. Knee-jerks absent on each side. No nystagmus, slight internal strabismus, probably congenital. The child had vomited several days ago, but there was no vomiting at the time of my visit. The temperature on admission was 101°, rectal, pulse 108. The case looked like one of tubercular meningitis, and was seen by Doctor Holt in consultation. Doctor Holt thought that on account of the slight elevation of temperature, and the absence of mastoid tenderness,—a tubercular process was the most probable explanation of the condition. He agreed with me, however, that in the presence of meningeal symptoms and a suppurating ear, in connection with the fact that aural suppuration had evidently been of longer duration than was suspected by the parents, as proven by the extensive amount of destruction in the drum membrane,—an exploration of the mastoid, with free exposure of the dura, would be advisable.

A complete mastoid operation was immediately done, and all the cells were found infiltrated with fluid. The dura was freely exposed. Over the temporo-sphenoidal lobe and over the left cerebellar lobe the dura was tense. The von Pirque test was negative. On the day after the operation the temperature fell about a degree, and the rigidity of the body diminished somewhat. On the following day, however, that is, two days after the mastoid operation, the rigidity became more marked, and the temperature rose to 103°. The pulse ranged from 116 to 150, and the respirations were rather high. Owing to the fact that the rigidity had increased, the child was taken to the operating room, and the cisterna magna drained in the median line, after the Haynes' method. The fluid was under considerable pressure. About three or four cubic centimetres of the spinal fluid, mixed with blood, was obtained for pathological examination.

This showed no germs, with the exception of one diplococcus. Immediately after the drainage of the cisterna magna, the child seemed to improve somewhat; the rigidity became less and the temperature gradually fell, until on the third day after operation, it was only 100°. It then, however, began to rise, and the child died seven days after the drainage of the cisterna magna. Some of the spinal fluid evacuated at the time of operation was injected into a guinea pig, and this animal remained perfectly healthy six weeks after inoculation, thus absolutely ruling out a tubercular process, as the cause of the infection.

The case, then, was one of a simple otitic meningitis, following a chronic middle ear suppuration, and should have been an ideal case for the procedure which we are discussing. That the life of the patient was prolonged by the operation, I believe. But the operation certainly failed to do more than postpone the fatal termination of the disease.

I might say that, in certain decompression operations, where the subdural space has been drained either in the cerebellar or middle cranial fossae, or in both regions, life has been prolonged for a number of days. I am inclined to think, however, in this case, that the time which elapsed between the drainage of the cisterna magna and the fatal termination of the disease, was rather greater than in most of my cases of decompression operations in the middle cranial fossa.

Case II. The second case was that of an infant, five months old, who was operated on by one of my assistants for a mastoid sub-periosteal abscess. This child was suffering greatly from malnutrition, and was a poor subject for operation. The temperature on admission was 102°; it fell spontaneously the next day to 100°, and then rose to 104°. An ordinary simple mastoid operation was performed. The temperature the day after rose to 105.5°, then dropped to 102°. Two days after the operation upon the mastoid the child had several convulsive seizures. The child was taken to the operating room and the cisterna magna opened in the typical

manner. The day after this operation the temperature rose to 106.5°, but then slowly came down, so that in the evening of that day it was only 100°, and the following day had dropped to normal. After remaining normal for about twenty-four hours, it began to rise and fluctuated between 103.5° and 100° until the child died, four days after the cisterna magna was opened. The infection in this case was streptococcus. The spinal fluid obtained by lumbar puncture was negative on cultivation.

In this second case the operation failed to be of the slightest value; in this instance, also, on tapping the cisterna the cerebrospinal fluid was found to be under considerable pressure. Drainage was perfect until the fatal termination.

Case III. The third case was that of a boy, aged eight, suffering from a chronic suppurative otitis media, for which the ordinary radical operation was performed by one of my assistants. A small area of dura was exposed in the middle cranial fossa. A primary skin graft was applied at the time of operation, and the posterior wound closed in the usual way. The temperature was normal at the time of operation, but immediately began to rise, and on the second day after operation, reached 105°. The posterior wound was opened and the graft removed, but the temperature still continued to rise. The blood culture showed one colony of streptococci at the end of twenty-four hours, but this was the only colony which showed at the end of forty-eight hours. In other ways, the blood was negative. The differential count showed 83° of polymorphonuclear cells on the second day after operation, and 74.8% on the fourth day after operation. The temperature, however, remained high, and the patient gradually sank into a semi-comatose condition. The neck became rigid, the Kernig sign positive; there was no Babinski. Lumbar puncture showed some pus in the cerebrospinal fluid, no germs present, and culture of the fluid was negative at the end of twenty-four hours. The carbohydrate content of the fluid was practically normal. Globulins were not present.

Owing to the fact that symptoms of meningitis were present, that the chemical examination of the cerebrospinal fluid was practically normal, and that the fluid was germ-free,—it seemed that the case was particularly suitable for drainage of the cisterna magna. This was performed four days after the radical operation. The fluid was under considerable pressure. The ordinary drain was inserted, and the patient returned to bed. The temperature remained high, but the patient seemed somewhat brighter for the next two days. He died, however, on the fourth day after drainage of the cisterna magna.

These three cases would seem to show that we have not yet discovered a surgical procedure which would lead us to combat successfully that dread disease, otitic meningitis. I do not mean to condemn the operation upon the results obtained in such a small number of cases. The operation is easy of performance, and certainly drains the subdural space as effectually as does any other procedure. It is possible that with more extended experience, this operation may be a step toward the solution of the successful treatment of otitic meningitis.

I have nothing to add regarding the technique of the operation, except to say that the elaborate armamentarium, suggested by Dr. Haynes, in his able article, is entirely unnecessary. I have been able to perform this operation with the ordinary mastoid instruments, and to perform it quickly. I believe that the procedure should be given a thorough trial, but the results obtained from my limited experience have certainly not been encouraging.

Discussion.

Dr. Ewing W. Day, of Pittsburg, Pa., had had twelve cases of septic meningitis during the past year which came to operation. The results were not encouraging. The time of life varied from twenty-four hours to twenty-one days. There was a short period of improvement, which seemed to depend upon the virulence of the infecting organism. In

the cases which lived twenty-one days there was mixed infection. There was a feeble growth only after twenty-two hours, this period of improvement being followed by sepsis. This cycle of sepsis seemed to run, without evidence of any pressure, until the case terminates in septic coma and death. The question arose, could anything be gained by drainage in these cases. There was one thing in common in all the post-mortem examinations: while the surface of the meninges was drained more or less completely, at the base of the brain there was an area which no drainage touched. There was a pool of pus in this locality which extended from the optic chiasm; over the pons, down the cord. The pus was confined to the anterior part while the posterior part was free. There was enough left in this pool after drainage, by every known method, to overcome any good that might be accomplished. Referring to Dr. Dench's case which improved under subdural drainage, he did not believe there ever was a case of general septic meningitis saved by subdural drainage. Subdural drainage influences only a very limited area. He did not believe decompression relieved the pressure at all. He emphasized the definite relationship between the cerebral circulation and the arterial and venous circulation. Pressure on the cerebral fluid must always be a little higher than that in the venous circulation and less than the arterial. As soon as this pressure is relieved it is immediately followed by an increase of fluid. The fluid will simply be increased that much more rapdly until the normal relationship is restored.

Dr. Wendell C. Phillips, of New York City, emphasized the fact that all that is hoped for by drainage of the cisterna magna is to relieve intracranial pressure. From his own experience, and from the few cases thus far reported, he was convinced that the operation does give relief of intracranial pressure, particularly if operation is resorted to early enough. He reported in this connection a case cited at the International Otological Congress in which there was streptococcus invasion as shown by the cerebrospinal fluid.

and in which the patient lived nine days, remaining fully conscious and in comparative comfort, after drainage of the cisterna magna. If there was any virtue in this operation at all it was in the prevention or relief of intracranial pressure; one must look elsewhere for means to tide the patient over the extreme sepsis. He was interested to hear Dr. Dench speak of pus without bacteria.

Dr. S. MacCuen Smith, of Philadelphia, had had three cases, two of which died on the table. The last was a child, six years of age, in which he had performed a mastoid operation. At the end of a week the temperature ranged around one hundred, and the nurse reported having noticed a twitching, she could not tell just where. He had not suspected meningitis on account of the child's good mental condition, but when he examined the cerebrospinal fluid he found an absence of the sugar reaction. The child died. At autopsy, in trying to separate the dura, there was a free flow of pus. A little higher up the cerebrospinal fluid was under pressure. This case illustrated the point he wished to make, viz., that it will require courage on the part of the otologist and of parents to consent to early operation where the child's mentality is good, even where the cerebrospinal fluid has been examined and sugar found to be absent. All were doubtless agreed that the operation does drain, except in cases where the pus is situated low down, as suggested by Dr. Day.

Dr. John D. Richards, of New York City, had seen one case in which, at post-mortem, it was found that drainage was not complete, fully half a cup of pus being discovered.

Dr. Joseph C. Beck, of Chicago, Ill., answering Dr. Haskin's criticism, said he had recently reported seven cases in which one lived. This was a pneumococcus infection in a girl, with secondary thrombosis, and ligation of the jugular. He operated early. Pneumococci and absence of sugar were found in the cerebrospinal fluid. The fluid was clear, and

the cellular elements were slightly increased. The girl made an uneventful recovery. It had been seven months since the operation. Four of the other cases were examined post-mortem, and in these the points made by Dr. Day was very clearly shown. Down along the tentorium, down to the foramen magnum, there was a yellowish exudate.

Dr. Frederick L. Jack, of Boston, Mass., cited a case operated upon by one of the assistant surgeons, in which it was thought that there was meningitis. A few days later a temporo-sphenoidal abscess was discovered. The patient recovered from the opening into the cisterna magna, as well as the operation on the brain abscess.

Dr. Dwyer said that in his opinion, we were wandering away from the true way of treating meningitis, which seemed to him to lie along bacteriological and specific antisera lines. Certainly every operation in the past, directed to the cure of meningitis by drainage had been a disappointment and after all, this operation is directed along the same lines. Considering the operation itself, there presented to his mind certain grave contra-indications, which may seem secondary in importance but which are important and these are all from the standpoint of the patient, as the operation itself is not difficult of performance. The position of the patient adds greatly to the congestion of the already congested meninges, a distinct contra-indication and also the use of ether, which is practically the anaesthetic that must be used and which is contra-indicated in such a condition. These are all small points apparently, but it is the small points that are the important ones in dealing with such a condition. After all, the only advantage gained is in the relief of pressure, as it is now well-known that the area drained is comparatively small, as proved at autopsy. Pressure could just as easily be relieved by lumbar puncture, as proved in the past. The cases that cannot be relieved by this operation also, as it is generally an internal hydrocephalus that is produced and the point of obstruction is

generally distant from the site of operation. Again, the cases the writer-speaker had seen in which the pressure symptoms were marked and which died in a few days died in spite of the relief of pressure and died with very marked symptoms of an acute toxaemia of fulminating type, and here the toxins were the important factor and not the mechanical pressure. Again we are all familiar with epidemic meningitis cases, in which the cerebrospinal fluid is very turbid, in whch the pressure symptoms are marked for days and in which complete recovery takes place after the use of the specific sera. In one case that the speaker recalls, the pressure had been extreme for forty-two days and yet recovery was complete. So we must look beyond the mere relief of pressure alone, and direct our energies to the combating of the toxaemia by therapeutic measures, such as sera, drugs, etc. During the past winter, we had a series of cases of meningitis due to the streptococcus mucosus capsulatus, in which we had absolutely no pressure symptoms at all and yet all died, without developing any of the symptoms we associate with meningitis. In this class of case therefore, the operation would not help at all. The speaker was fortunate enough to have a case of otitic meningitis recover completely, in which the organism, the streptococcus, was isolated from the cerebrospinal fluid. In this case the Hiss Extract of leucocytes and hexamethylamine were used: the symptoms of pressure were marked for four or five days but cleared as the spinal fluid cleared. In his opinion therefore, more was to be hoped from the treatment along therapeutic lines than from this operation.

Dr. Dench, in closing the discussion, referred again to the first case, which had been criticised. All who saw the patient thought it was a case of tuberculous meningitis, but it could not be proved by any test. The guinea-pig test, which pathologists agree is the best, failed in this case. He did not think there was any more evidence in favor of tuberculosis than against it. Dr. Day's work carried with it so much weight that he hesitated to speak of it. In

a case of meningitis just beginning, with infection through the tympanic roof, if one drains good results will be obtained. The statement that drainage is limited is true, but drainage of the labyrinth is also limited. Dr. Day had struck the keynote of success in the surgical treatment by emphasizing the fact that operation must be resorted to early. The indications seemed to him to be for wider drainage, that is, drainage in several places. He had tried urotropin, according to the suggestion of Dr. McKernon, and the cases died just as before. He had had the same experience with antistreptococcic serum. He did not consider the Haynes operation dangerous or difficult. Cases of otitic meningitis in some instances get well by various measures, some by lumbar puncture, some by sera, etc. The only way to determine the most satisfactory method of procedures was by each man reporting his cases.

EXPOSURE AND CURETTEMENT OF THE ATTIC COMBINED WITH A MODIFIED BLOOD CLOT AS FACTORS IN PROMOTING RAPID MASTOID HEALING.

By HUGH B. BLACKWELL, M. D., New York, N. Y.

The following paper is based upon the study of sixty-nine (69) cases of mastoiditis operated upon during the past four years, in all of which a form of blood clot dressing was used. The cases were undertaken for the purpose of reducing the time of healing, diminishing the pain of dressing, and improving the appearance of the scar, subsequent to mastoidectomy.

When a form of blood clot healing has been attempted and results in failure, the cause lies obviously in the infection of the clot. This infection may arise from within the wound or be introduced from without. Owing to the surgical pathology of mastoiditis, it is safe to say that in the vast majority of instances the infection of the clot, when it takes place, occurs from within the wound. If a thorough operation has been performed, there should be only one source of infection in the wound sufficiently gross to always seriously menace the integrity of a clot; namely: the tympanum—the original focus of the disease. In thirty-eight (38) cases of this series, in which the attic of the middle ear was exposed and curetted, this region was found completely filled with infected granulation tissue. By attic, we refer to the upper portion of the tympanic cavity lying above the level of the horizontal facial canal and the epitympanic ring of bone containing the body and short process of the incus and the head of the malleus. Owing to the proximity of the clot to the infected granulations in this region, it is evident that if the infected tissue were not recognized and removed at the time of the operation it would be sufficient to cause frequent infection and subsequent failure of the blood clot when employed. When a form of blood clot healing is attempted it

is most essential that every evidence of disease is removed from the mastoid and in addition as much of the infected tissue from the attic of the ear as is possible without a disturbance of the ossicular chain.

Operation.

When all macroscopical evidences of diseases have been removed from the mastoid bone, including the antrum and zygomatic regions, the tip is removed and the posterior bony canal wall taken down to within about one-eighth to one-quarter of an inch of the epitympanic ring of bone. The cap of the external semi-circular canal is exposed and the granulation tissue lying in front of and below it is cautiously removed revealing the short process of the incus imbedded therein. These granulations are removed from around the incus, freely exposing its short process and body. A small narrow curette is inserted in front of and external to the incus and the external attic wall curetted away by working from within outward. After removing the granulations from the external attic the head of the malleus becomes visible. With a small narrow curette the granulations are removed from the internal attic. In order to safely expose the ossicles and curette the attic it is necessary to remove the external attic wall. Operative work upon the cadaver has shown us that if the ligament which binds the end of the short process of the incus to the bone below and in front of the external semi-circular canal is ruptured while curetting around the ossicles, manipulations in this locality had best be concluded at once as further efforts would probably dislocate that ossicle. It being its main ligamenlous support. The ligaments of the malleus are much more easily avoided, as the operator, in taking down the external attic wall always works above the external ligament of the malleus, and in curetting the external and internal attic the curette is used on either side of the superior ligament of the malleus. A few drops of adrenalin blanches the attic and renders the ossicles more visible. Loose bone chips should be removed as soon as they accumulate in order to prevent

their falling into the tympanum. Care should be taken not to tear into the external auditory canal by undue efforts at retraction. At the conclusion of the operation, the external auditory canal is packed snugly with plain gauze. The canal is kept packed for at least two weeks after the operation, thus preventing the collapse of the posterior membranous canal wall, which is now of course devoid of bony support. The amount of blood used to fill the wounds varies considerably in each case. In some the mastoid wound is packed lightly, only permitting a shell of clot to form around the gauze, while in others just a strip of gauze was laid down to the antrum. In only three cases was there no posterior packing employed. The other cases, sixty-six (66) in all were modified by more or less gauze packing which was placed in the posterior wound at the close of the operation.

The wound is sutured down generally to within the last one-half inch and a wet dressing of normal saline applied.

In the first thirty of these cases a specialized form of dressing was used. This has been discarded. The sutures are usually removed at the end of forty-eight hours at the time of the first dressing. After this the dressing is changed daily until all of the serum has been squeezed out of the clot in its process of organization. The posterior packing may be discontinued after the second or third dressing, depending upon the amount of discharge present, etc. When the wound shows any tendency to suppurate it is dried out and packed lightly with iodoform gauze. The dressing is usually kept wet for the first week.

Resume.

Of the sixty-nine cases, forty-two were adults, seventeen were between one and sixteen years of age, and ten were babies under one year. The twenty-four known infections consisted of thirteen streptococcus, 3 streptococcus capsulatus, 3 pneumococcus, and two mixed infections. The twenty-first day after the operation, thirty-two of the cases were entirely healed and all dressing removed. Of this number twenty were adults, 8 were children under sixteen years

of age, and 4 babies. At the end of the twenty-eighth day subsequent to the operation 12 of the remaining cases were entirely cured, of which number 7 were adults and 5 were children between one and sixteen years of age. At the end of the thirty-fifth day 6 of the remaining cases were declared cured, 2 were adults, 2 children and 2 babies. Excepting 3 all of the remaining cases were cured at the end of the forty-second day. One adult who required four and one-half months to heal, another adult who is unhealed at the end of five months, who had a marked dyscrasia at the time of the operation, and a child one and one-half years old at present unhealed eight weeks after the operation. Of the entire series of sixty-nine, there are 13 cases whose exact date of healing was not noted, and are not included in the above statistics. I can say, however, that their convalescence about corresponded in point of duration to that of the other cases. Nineteen of the cases were perisinous epidural abscesses, 23 were sub-periosteal abscesses. Nine had chronic discharge from the ear, there being little or no polypoid granulations present in the tympanum save in the attic, and the hearing being good, the attempt was made to save it. Three of the cases were sub-periosteal abscesses opening through the posterior bony wall of the external auditory canal and were first diagnosed and treated as feruncles. In each instance they were complicated by perisinous abscesses. Three developed erysipelas. They were all mild cases. In all of the three instances the wounds were doing well at the time of the development of the complication which first appeared some distance away on the face. Dura or sinus were exposed in 32 of the cases. None of the cases in this series died, and in only one was there an operation for recurrent mastoiditis which developed some two years after the primary operation. In none of the cases did the dislocation of an ossicle occur.

Quantitative functional tests conducted after recovery have shown that the hearing was no more impaired in the cases in which the attic was curetted than in the previous ones in which the attic was undisturbed. The external auditory canal

will not collapse if packed snugly at each dressing. It is quite interesting to observe the condition of this packing when removed from the auditory canal at the occasion of the first or second dressing, as it is nearly always absolutely dry, which signifies that the infection had been removed from the tympanum at the time of the operation.

Conclusions.

We believe that in a certain number of selected cases of chronic middle ear discharge with good hearing, presenting evidences of true attic suppuration only, that this operation combined either with or without a blood clot, or with or without a plastic meatal flap, will preserve the hearing and remove from the ear its potential possibilities of menace, and

That when the procedure is employed in acute mastoiditis the duration of healing after mastoidectomy can be very materially shortened, that the dressings are less painful, the convalescence more comfortable, and the subsequent scar presents a much better appearance.

Discussion.

Dr. Edward B. Dench, of New York City, thought the modified blood-clot operation of distinct value. While the modified blood-clot did not entirely close the wound, better results were obtained by this means than by keeping the wound entirely open.

Dr. William C. Braislin, of Brooklyn, N. Y., thought the modified blood-clot method unsatisfactory in young children because of their tendency to get their fingers under the dressing and thus more or less infect the wound.

Dr. Wendell C. Phillips, of New York City, protested against the extreme and unnecessary radicalism described in this paper. He would personally not submit to the radical method advocated by Dr. Blackwell.

Dr. Christian R. Holmes, of Cincinnati, Ohio, agreed with Dr. Phillips in objecting to placing the stamp of approval

of the society upon this paper and upon the very radical measures which its author advocated. He considered it a dangerous procedure to curette around the ossicles, as suggested by Dr. Blackwell. As to the blood-clot method, he considered this a matter of technic. He had been a champion of the method, and still used it in some cases.

Dr. John D. Richards, of New York City, had seen Dr. Blackwell operate in a large number of cases, and it so happened that a few years ago he had himself presented a thesis before the American Laryngological, Rhinological and Otological Society in which he advocated exposure of the atticus tympanicus. He thought Dr. Phillips and Dr. Holmes in error in placing a stigma upon a paper of this character, and believed they would not do so if they saw Dr. Blackwell operate and watched his results.

Dr. Philip D. Kerrison, of New York City, had had occasion to follow Dr. Blackwell's work and had found his results particularly good. The drum membrane was clean very early, and healing behind the ear was perfect and very rapid. There was nothing, in his experience and observation, to warrant condemning the operation.

Dr. Blackwell, in closing the discussion, said he was glad to hear the criticism with reference to the work around the attic. The paper was entirely clinical, representing work performed at the Manhattan Eye and Ear Hospital and the New York Eye and Ear Infirmary. The reason for curetting the attic and combining with the blood-clot dressing, is that the cases heal quickly. There is no subsequent interference with hearing. The paper was based upon sixty-nine operations, in thirty-nine of which the attic was curetted. These cases did well. The justification for cleaning out the attic was that when the modified form of blood-clot was to be used it was necessary to get the wound as clean as possible. As much infection must be removed from the middle ear at the time of operation as possible, otherwise the wound would be more apt to become infected and break down.

A REPORT OF THE RESULTS OF SEVEN CASES OPERATED UPON BY MR. HEATH.

By EDWARD M. PLUMMER, M. D., and HARRIS P. MOSHER, M. D., Boston, Mass.

INTRODUCTION.

A week before the Ninth International Otological Congress held in Boston in August, 1912, Mr. Charles Heath of London was given the freedom of the Aural Department of the Massachusetts Charitable Eye and Ear Infirmary. This was done through the courtesy of the board of trustees and of the surgeons then on duty. Mr. Heath was given his choice of cases, and selected seven upon which he operated with his own instruments and with the help of his own assistant. He did the dressings and directed all the treatments during his stay, that is, for about two weeks. During this time he was most gracious and painstaking in instructing the surgeons on duty and the house staff in his method of operating and in his mode of after treatment. At his departure, he left at the hospital a set of instruments for carrying out the after treatment and the formulae of the solutions used for this purpose. During the remainder of August, and for the whole of September and October, in other words, for the rest of the service, Mr. Heath's cases were supervised and dressed by Dr. Plummer and Dr. Mosher, occasionally spelled by the senior house officer, Dr. MacMillan, who was second assistant at the operations and had followed the cases from the start. When the cases were discharged from the house, and they were not discharged until the end of the service, they came under the care of the assistant surgeon in the Out-Patient Department. At first, Mr. Heath's method of after treatment was followed rigidly. When certain cases failed to respond to this, the usual after treatment of the hospital was substituted. What

this was can be found in the detailed history of the cases. On April 26, of this year, roughly seven months after the operations, a letter was sent to all of the patients asking them to report for a final examination. Four of the cases reported, one was out of town and will report later; two cases could not be located.

The subject of this paper is the results obtained by Mr. Heath in the seven cases which he operated. The steps of his operation and his method of performing them are in print and will not be discussed. Further the paper does not deal with his reasons for considering the cases operable. A little is added, however, on the progress of the cases during healing. The writers of this report are in a position of considerable delicacy. They are reporting the results of the work of a guest; a guest who did not have the opportunity of following his cases and who cannot be present at the discussion of them. He was untiring in demonstrating and explaining the cases to us, and then left them manfully and unreservedly in our hands. They are reported without obtaining his formal consent. The report, of course, will be sent to him. Any man can see that a spirit of fairness is necessary on both sides. There was a tacit understanding, that Mr. Heath wished a fresh, and a candid and impartial consideration of his work. He paid us the compliment to come to this country for it. We have tried to give it. The writers hope that the bitter personalities which characterized the debate on Mr. Heath's work last August will not be repeated.

The report is made up as follows: The detailed histories of the cases just as they stand in the hospital records are first given, abbreviations and all, without change. This part will be skipped in the spoken paper. The reader will do well to take this part of the paper last. After the detailed histories are given, a short summary of each case is presented. The writers comment on each case, and then summarize the abstracts. The last few paragraphs give some of the impressions which the writers of the report gained from watching Mr. Heath operate and from following the cases afterwards. The detailed histories of the cases follow:

PART I.

Vol. 34, page 301. Margaret Donovan. Age 6. Residence, 21 Carson street, Dorchester, Mass. Birthplace, Massachusetts. August 12, 1912. Re-entry Jan. 19, 1912. Vol. 33.

Diagnosis: A. S. o. m. s. a. A. D. o. m. s. a. Recurrent mastoiditis—post aural abscess.

Operation: Heath Conservative mastoid. Mr. Heath.

History: A. D. patient was admitted to this hospital Jan. 19, 1912, suffering from A. S. o. m. s. a. A. D. mastoiditis with post aural abscess. A mastoid operation was done by Dr. Lothrop, in which the abscess was incised and the cortex found perforated near canal. Cortex removed and all softened cells curetted out. Antrum found and curetted. Sinus exposed but not opened. Discharged on Feb. 1, 1912. A. D. well until last Monday (one week ago) when pain commenced and has lasted to date. There has been discharge for the past three days. Mastoid tenderness has existed for four days, and swelling for three days. P. E. negative.

Examination: A. D. mastoid scar of previous operation. Marked post aural redness, bulging and fluctuation. Small round perforation in lower part of M. T. Very profuse purulent discharge. A. S. M. T. red and bulging. No landmarks seen. Under primary ether M. T. incised.

Aug. 13. Operation. Ether. Mr. Heath. Usual Heath incision. Pericranial flap cut. About ʒ ss of pus escaped from under periosteum. Small pin head perforation found in reformed lateral wall of mastoid. Antrum exposed from front and posterior canal wall removed. Antrum burred out and meatial flap cut and M. T. exposed. Perforation in posterior half of drum. Two flaps sutured together. Dressing to antrum and meatus. Usual cut made through helix and tube inserted. Wound closed with seven s. w. g. sutures. Moist dressing. Bandage.

Aug. 14. Sutures removed. Moist dressing reapplied.

Aug. 19. Wound dressed daily by Mr. Heath, who has changed the tympanic and antral dressing daily.

Aug. 25. Incision firmly healed behind. Incision through crus of helix gaping and granulating. Considerable mucous about tube still. Antrum granulated and full. Exuberant granulations on bridge and on post. sup. segment of M. T. Single gauze wick to M. T. Superior wall of meatus projecting. Tube left out and gauze plug inserted instead.

Aug. 29. Skin incision firmly healed. Scar of first operation slightly elevated. Incision of present operation scarcely visible. Incision through helix healed without deformity. Wick well saturated with pus. Antrum granulated full. Flap in place. Pus in M. E. Large granulations over upper anterior border of bridge, hiding most of M. T.

Sept. 6. Moderate amount of secretion on wick. Antrum closed. M. T. $\frac{2}{3}$ seen. Moderate muco-purulent secretion coming from M. E.

Sept. 18. Small area of granulation on bridge removed. Discharge continues.

Sept. 28. Discharge from M. E. has stopped.

Oct. 3. After remaining dry for two days discharge noticed again but only slight amount.

Oct. 7. No discharge from M. E. since last note.

Oct. 10. Discharged relieved to O. P. D.

Vol. 34, page 258. Name, Joseph Weinstein. Age, 21. Occupation, factory operator. Birthplace, Russia. Residence, Eastman House, Revere, Mass. July 29, 1912.

Diagnosis: A. D. o. m. s. a. Mastoiditis.

Operation: Heath mastoid operation. Mr. Heath.

History: A. D. patient had a "head cold" three weeks ago, following which A. D. pained for one day and was lanced by Dr. Myers of Chambers street. A. D. has discharged since and patient has syringed it. Pain stopped until two days ago, when it became very severe at intervals. He has had occasional headaches. Mastoid tenderness two days. P. E. negative. Urine negative.

Examination: Considerable tenderness over tip and antrum. No post aural redness. Very profuse purulent discharge from M. E. Large irregular perforation in posterior lower quadrant. Considerable œdema of superior canal wall.

Aug. 3. Operation. Ether. Mr. Heath. Incision made at junction of attachment of auricle posteriorly. Pericranium exposed and temporal muscle. Pericranial flap cut and held back, cortex exposed. Antrum exposed from the front, cortex being removed along anterior part of bony canal. Antrum burred out. Meatial flap cut and M. T. exposed. Perforation found at lower posterior quadrant. Posterior canal wall previously having been taken down to within ⅛ inch of M. T. Flaps trimmed and sutured with one C. G. suture. Canal incised through helix. Dressing to antrum and tympanum. Tube fitted and inserted into canal. Wound closed with 7 s. w. g. sutures. Moist dressing. Bandage. Lotion washed out many small white flakes, said to be cholesteatoma. (Mr. Heath.) Air forced from antrum through perforation in M. T.

Aug. 4. Sutures removed. Moist dressing reapplied. No temperature. No headache.

Aug. 5. Tube removed from meatus. Dressing removed from antrum. Dressing very painful. Plug left in meatus. Moist dressing reapplied.

Aug. 6. Under gas plug removed from antrum. Antrum wiped with iodoform emulsion and small dressing reapplied to antrum and tympanum.

Aug. 7. Tube changed. Antrum and tympanum redressed with plugs wet with iodoform emulsion. Air blown through perforation by Valsalva's method.

Aug. 10. Temperature up to 103°. Headache for three days. Mastoid wound well healed.

Aug. 14. Up and about. Dressed and tube changed every day since operation.

Aug. 19. Incision fully healed. No induration of wound. Temperature 99°. Headache during P. M., beginning at 4 P. M. for several days. No pain in ear. Slight discharge. Incision through crus of helix ¾ healed. Slight granula-

tion in lower part. Considerable discharge from meatus. Few granulations on upper meatial wall along edge of flap. Perforation patent by Valsalva. Tag of flap on upper wall.

Aug. 22. Profuse amount of discharge from M. E. Incision through helix healed. Tag of flap on superior wall still. Antrum ½ obliterated. Bridge covered with granulations. Valsalva successful. Pus blown from M. E.

Aug. 26. Considerable amount of granulations from incision through helix, these were trimmed and cauterized with Ag No_3 stick. Antrum well filled in. M. E. still discharges freely. Valsalva shows patent perforation and semi-purulent discharge blown through.

Aug. 30. Incision through crus of helix slightly gaping inferiorly, otherwise healed. Moderate amount of discharge from canal. Valsalva successful, expelling pus from M. E. Antrum granulated full. Grans on anterior surface of bridge and covering in places the M. T. Slight tag of flap in middle of superior wall. Gauze wick saturated with zinc. sulph. to M. T. Tube reapplied.

Sept. 6. Tube still in. Much foul discharge about tube. Incision through crus of helix healed without deformity. Much pus in canal and about M. T. Slight dimpling at site of antrum. Flap well back and in place. No tags. Thin pus blown from M. E. Posterior half of M. T. seen. Tube out, gauze wick to canal.

Sept. 27. Tag of flap posteriorly superiorly. Canal filled with aristol. Two days ago patient developed cervical adenitis on right side of neck. R Hot sulpho-naphthol poultices.

Oct. 4. Neck continues to be painful. Swelling less. Discharge, however, continues from M. E., but now only slight amount.

Oct. 10. Ether. Operation. Dr. MacMillan. Right tonsil removed in usual way. No point of infection found. Left tonsil not touched, as patient did very poorly under ether and tonsil presented no indication for removal.

Oct. 16. Since removal of tonsil adenitis has improved, but ear still discharges a small amount of pus daily.

Oct. 21. Adenitis well. M. E. still slightly moist. Two-thirds of M. T. visible. Antrum well epidermatized. Small round perforation in lower posterior quadrant. Discharge relieved to O. P. D.

Dec. 30, 1912. A. D. M. T. of normal appearance; landmarks seen; antrum well epidermatized over. Ear dry.

August 1, 1912.

R				L
18				18
—		W		—
25				25
—		V		—
8		AC	25″	15
—	F 512 V. S.	—	—	—
10		BC	12″	10

WEBER < F 256 V. S.

N	LIMITS GALTON	N
128 V. S.	LOW	V. S. 80

September 21, 1912.

R				L
15				18
—		W		—
25				25
—		V		—
12		AC	25″	21
—	F 512 V. S.	—	—	—
14		BC	12″	12

WEBER < F 256 V. S.

N	LIMITS GALTON	N
128 V. S.	LOW	V. S. 80

October 21, 1912

R				L
18				18
—		W		—
25				25
—		V		—
23		AC	25″	22
—	F 512 V. S.	—	—	—
14		BC	12″	18

WEBER < F 256 V. S.

N	LIMITS GALTON	N
80 V. S.	LOW	V. S. 80

Vol. 34. Page 273. Frank Sapanari. Age, 30, Birthplace, Italy. Residence, Attleboro, Mass. Laborer.

August 31, 1912.

Diagnosis: A. D. o. m. s. a.

Operation: Heath Conservative Mastoid.

History: A. D. Two months ago the patient had pain below the right auricle. It soon went away and one month ago he felt the ear blocked up. He came to O. P. D. and had cerumen syringed out of both ears. At that time he had otitis externa diffusa in A. D. and on July 2nd, 1912, canal was incised in O. P. D. Ear commenced to discharge 6 days ago, and mastoid became tender 5 days ago and has been so since. P. E. negative.

Examination: A. D. external canal filled with polyp. Purulent discharge from M. E. No part of M. T. can be seen. Marked mastoid tenderness over antrum, tip and emissary.

Aug. 4. Under gas polyp was removed from A. D. to give better drainage.

Aug. 8. Patient examined today by Dr. Emerson with naso-pharyngoscope. Sharp spur on right side of septum in contact with turbinates. Large posterior ends. Vault congested especially on right.

Aug. 9. Operation. Ether. Mr. Heath. Usual Heath incision, etc.

Aug. 10. Sutures removed. Dressing reapplied.

Aug. 15. Patient has been dressed daily by Mr. Heath, who has changed tube and antrum and tympanic dressing and applied iodoform emulsion daily.

Aug. 20. Middle ⅓ of incision not quite healed. Granulation trimmed at this point. 1 c. m. of incision at upper extremity healed.

Sept. 6. Incision through crus of helix completely healed. Slight redundancy of tissue with slight eversion of posterior lip. Flap boggy. Antrum half granulated in. Considerable pus in M. E. Valsalva successful. Pus expelled. Antrum one-half obliterated. One-fourth of lower posterior part of M. T. seen. Zinc sulp. continued.

Sept. 27. Canal narrowed slightly externally. Superior ridge of flap projects. Opening into antrum 2 m. m. in diameter showing pulsating discharge from M. E. M. E. hard to distinguish. Profuse purulent discharge until last three days. ℞ Sol. M. 2 i. d. Ag No_3 bead to granulations.

Oct. 10. For past few days discharge from M. E. has been less than before. Antrum, however, has failed to fill in.

Oct. 18. Only slight discharge from M. E.

Oct. 25. Slight moisture continues from M. E. Antrum not filled in.

Oct. 29. For past few days practically no discharge from M. E.

Nov. 1. Discharged, relieved to O. P. D.

Patient has been seen from time to time in the O. P. D. (by A. L. M.). Last seen on Dec. 20th, when M. E. was dry and considerable part of antrum filled in. M. T. seen but no perforation noted. Landmarks made out. H. T. 18/25; Rinné 14/9; Low limit 96.

August 3, 1912.

R				L
1/6				18
—		W		—
25				25
—		V		—
5		AC	25″	10
—	F 512 V. S.	—	—	—
11		BC	12″	15

WEBER < F 256 V. S.

N	LIMITS GALTON	N
128 V. S.	LOW	V. S. 80

Vol. 34, page 305. Louis Kaplan. Age 2. Residence, 161 Union street, Rockland. Aug. 14, 1912.

Diagnosis: A. S. o. m. s. a. Mastoiditis. Post aural abcess.

Operation: Heath Conservative Mastoid.

History: Patient has had discharge from A. S. for past ten days. Swelling developed over mastoid region six days ago. P. E. negative.

Examination: A. S. Post aural redness, oedema, and tenderness. Well localized post aural abscess. M. T. red and bulging posteriorly. No perforation seen. Considerable purulent discharge from M. E.

Aug. 15. Operation. Ether. Mr. Heath. Usual Heath incision, etc.

Aug. 19. Up to today, wound has been dressed daily by Mr. Heath. Sutures removed day after operation, since then antrum and tympanic plug changed daily. Moist dressing used.

Aug. 25. Antrum filled with granulations. Gauze wick to M. T. Owing to gaping of canal wound no wick inserted.

Aug. 30. Skin incision healed. Incision through helix gaping. Much discharge. Edge of canal flap projecting. Antrum granulated full. Flap boggy throughout, hiding M. T. Gauze wick to meatus.

Sept. 6. Incision through crus of helix healed with 2 m.m. separation of edges. Much pus from canal. Flap projecting into canal posteriorly readily pushed back, and large granulation seen at tip of flap projecting forward, hiding antrum and M. E. Granulation removed from tip of flap.

Sept. 20. Flap has been touched up daily with Ag No_3 bead and now able to see M. E. Discharge less.

Oct. 1. Canal now free with only slight projection of flap. Very little discharge from M. E.

Oct. 5. Profuse discharge continues from M. E.

Oct. 16. Discharge as at last note. Today at lower half of mastoid incision there is considerable redness with slight purulent discharge.

Oct. 21. Posterior wound has quieted down. Only slight discharge from wound. Discharge continues from M. E.

Oct. 29. Discharge still continues.

Oct. 30. Middle ear discharge continues and condition of mastoid wound remains the same.

Nov. 5. Re-infection of mastoid wound fails to respond to treatment.

Nov. 9. M. E. still discharging. Bleeds easily. Few granulations seen. Scharlach red to M. E. Mastoid wound unhealed yet.

Nov. 12. Mastoid wound and middle ear still discharging a good deal. ℞ Tinct. iodine to both.

Nov. 14. Amount of Scharlach red reduced. Mastoid wound red and tender. M. E. still discharges.

Nov. 16. Tinct. iodine to mastoid and middle ear. Both discharging a good deal.

Nov. 20. Wound back of ear discharges less, but fails to heal. M. E. discharges a good deal still.

Nov. 24. Wound over mastoid still fails to heal and middle ear discharges yet.

Nov. 27. Condition the same.

Dec. 2. Posterior wound healing a little. M. E. discharging a good deal yet.

Dec. 5. Operation. Ether. Dr. Jack. Incision made close to auricle to include old incision. Periosteum re-

flected. Small mastoid cavity found below leading to antrum. Cavity enlarged and curetted. Antrum and M. E. cleaned of granulations. Malleus removed. Eustachian tube curetted. Cavity smoothed out, and posterior canal wall removed to facial ridge. Small graft cut from right thigh and placed within cavity, being held in place by mild iodoform tape. Wounds closed with 5 s. w. g. sutures. (Flap was cut from posterior canal wall).

Dec. 7. Patient's temperature has been up since operation, but he appears to be bright and does not complain.

Dec. 10. Dressing changed. Considerable discharge from M. E. and mastoid. Stitches removed, as they had sloughed. Temperature still up, somewhat elevated. Leg was dressed yesterday, is doing well.

Dec. 12. Patient's temperature is still up a little. Posterior wound failed to heal over, and packing projects through it. There is considerable discharge, causing some excoriations of the auricle.

Dec. 14. Packing removed from middle ear. Posterior wound open for about 3 c. m. and gaping. Both M. E. and mastoid wound discharge profusely. Cavity filled with granulations. Evidently graft has not taken.

Dec. 20. Radical cavity dressed daily. Evidently more of graft took. Cavity is covered with granulations and considerable discharge persists from cavity and posterior wound, which is gaping for about 3 c.m. Cavity painted daily with scarlet red 4% in oil.

Dec. 26. Cavity cleaner and wound posteriorly is slowly closing in. Cavity is beginning to show considerable area of epidermatization, especially posteriorly.

Dec. 31. Posterior wound slowly closing in. Considerable discharge continues from cavity. Daily dressings continued. Boric powder in large amounts is now being used.

Jan. 4. Wound slowly improving. Daily dressing with boric powder continued. Considerable area of radical cavity has now epidermatized over.

Jan. 10. Mastoid wound still open and discharge quite

abundant. Cavity is epidermatizing and there are not any grans to be seen. ℞ pulv. boric.

Jan. 16. Less discharge from mastoid wound, which is closing up slowly. M. E. still discharges a good deal.

Jan. 18. Discharged improved to O. P. D.

Vol. 34, page 224. Name, Russell Noyes. Age, 12. Birthplace, New Hampshire. Residence, 35 Fifth street, East Cambridge, Mass. July 19, 1912.

Diagnosis: A. S. o. m. s. ch.

Operation: Heath Conservative Mastoid. Mr. Heath.

History: A. S. patient has had discharge from A. S., about once a year with pain at times, and deafness for the past six years. P. E. negative. Urine negative.

Examination: A. S. large perforation involving posterior half of M. T. Inco-stapedial joint exposed and covered with granulation tissue. Considerable purulent discharge. A. D. M. T. atrophic.

July 29. Under local treatment ear has shown no signs of improvement. Seen by Dr. Knowles, who advises patient to wait for Mr. Heath.

Aug. 12. Operation. Ether. Mr. Heath. Usual Heath incision. Pericranium exposed and flap cut. Flap held back with clip and cortex exposed. Cortex removed at junction of mastoid wall and bony canal and antrum exposed from front. Posterior canal removed down to anulus and M. T. exposed, showing large perforation in posterior half. Antrum well cleaned out with burr. Dressing to antrum and tympanum. Wound closed by 7 s. w. g. sutures. Large tube inserted through incised helix—moist dressing—bandage.

Aug. 13. Sutures removed. Dressing changed.

Aug. 18. Wound has been dressed daily by Mr. Heath, who has changed antral and tympanic dressing daily, and tube.

Aug. 20. Gauze stained moderately with pyocyaneus. Incision firmly healed in upper half, not quite firm in lower

half. Small area of granulations in middle. Abundant purulent discharge around tube. Incision through crus of helix slightly gaping and not healed. Some loss of substance about incision in helix. Antrum, bridge and posterior third of M. T. easily seen. Valsalva not successful. Bridge bare of granulations.

Aug. 22. Antrum one-third filled with granulations. Bridge two-thirds covered. Incision not healed. Flap in good position. Cotton plug of iodoform emulsion to antrum. Gauze wick to M. T.

Aug. 28. Upper two-thirds of incision firmly healed. Lower third slightly gaping. Very little discharge about tube. Incision through crus of helix healed. No deformity. Superior border of flap slightly projecting along median line of superior wall. Antrum one-third obliterated. Valsalva negative. M. T. hidden by granulations from bridge. Bridge covered with granulations. Gauze wick with zinc. sulph. to M. T. Wick to antrum.

Sept. 6. Incision practically healed. Incision through crus of helix firmly healed with dimple at upper end. No noticeable deformity. Gauze wick moderately stained. Flap firmly united posteriorly and superiorly. Large granulation over bridge hiding antrum. M. T. not seen. Valsalva negative. Granulation removed from bridge. Antrum then found to be filled with soft granulations.

Sept. 27. Antrum granulated in and ready to epidermatize. Sero purulent discharge from M. E. Anterior half of M. T. red and boggy. Scarlet red to cavity and Sol. M. t. i. d.

Oct. 1. Condition of ear about the same as at last note.

Oct. 13. Today only slight moisture from M. E. Granulation over bridge touched with Ag No_3 bead.

Oct. 19. Antrum closed except for small area about 4 mm. in diameter and of same depth. Very slight moisture in M. E. Posterior half of M. T. seen and rest covered with granulation tissue.

Oct. 21. Discharged relieved to O. P. D.

Dec. 28, 1912. Considerable discharge—slight moisture in antrum, but antrum well epidermatized.

December 28, 1912.

R				L
18				15
—		W		—
25				25
—		V		—
36		AC	25″	17
—	F 512 V. S.	—— —	——	—
21		BC	12″	14
	WEBER < F 256 V. S.			
N	LIMITS GALTON			N
96 V. S.	LOW			V. S. 96

Vol. 34, page 275. Name, Murphy, Daniel. Age, 13. Birthplace, Cambridge. Residence, 21 Frank street, Cambridge, Mass. Aug. 5, 1913.

Diagnosis: A. S. eff. o. m. s. Acute exacerbation.

Operation: Heath Conservative Mastoid. Mr. Heath.

History: A. S. patient has had discharge from A. S. at times since infancy. Three weeks ago pain started. Mastoid tenderness developed this A. M., but there is no swelling. History of similar attack one year ago. P. E. negative. Urine negative.

Examination: Profuse purulent discharge from M. E. M. T. red, but not bulging. Small round perforation in posterior lower quadrant. Patient unable to blow air through perforation. Moderate tenderness over antrum, none over tip. No redness or oedema.

Aug. 9. Patient has remained about the same since admission. Seen several times by Mr. Heath, who advised operation. Tenderness less. Profuse discharge from M. E. Transillumination by Dr. Mosher shows slightly light antrum and dark tip.

Aug. 10. Operation. Ether. Mr. Heath. Cortex exposed in usual way. Pericranial flap cut. On opening cortex pus continued to discharge for some time. Probe passed ⅔ inch upward into antrum and for same distance downward to tip. After pus, blood flowed for some time from the vascular mucous membrane of antrum. This hemorrhage caused

slight delay. Antrum treated in usual way with burr. Tip exposed and most of outer wall of a large tip cell removed. Granular tissue in cell left. Small wick drain at tip. Wound closed with one s. w. g. suture.

Aug. 12. Sutures removed. Tube changed.

Aug. 14. Patient dressed daily by Mr. Heath. Tube changed and antrum and tympanic dressing changed daily.

Aug. 18. Incision slightly gaping for full extent. Slight amount of secretion expressed from top. Base of flap which bounds meatus posteriorly and just below helix turned outward, leaving small granulating area. Antrum still large M. T. not seen. Valsalva negative.

Aug. 20. Gauze moderately stained with pyocyaneus. Incision slightly separated in upper one-half and whole of incision uncaught. Much muco-purulent secretion about tube. Abundant granulations on edges of incision through helix, also on posterior half of meatus. Antrum still large. One half of bridge covered with granulations. Valsalva successful. Moderate amount of watery discharge blown through. Posterior third of M. T. seen.

Aug. 28. Upper third of incision granulating. Lower third of incision granulating. Incision through crus of helix healed. No deformity. Moderate amount of pus about tube. Wick well saturated with pus. End of canal flap slightly projecting. Roof slightly projecting. Antrum only slightly granular about edges. Bridge covered with granulations except superiorly. Small rim of granulations on bridge anteriorly covering one-third M. T. Valsalva successful. Muco-purulent discharge blown from M. E. Rise of temperature which occurred two days ago has subsided. Gauze wick to antrum and tympanum. White blood count 20,000.

Sept. 6. Incision in lower two-thirds healed. Upper one-third gaping. Flap well in position posteriorly. Superiorly the canal wall falls a little. Antrum apparently very little granulated in. Bridge covered with granulations. Valsalva negative. Rim of granulations extends from bridge and obscured upper half of M. T. Granulations removed

from posterior part of bridge. Plain gauze to antrum. Zinc sulphate to M. T.

Sept. 27. Since last note patient has been dressed by Dr. Plummer. No tag or flap in region of antrum, hole 4 cm. in diameter into which probe enters 1 cm. Area of granulations on bridge posteriorly, superiorly. Removed and site touched with Ag. No. 3 bead.

Oct. 4. M. E. still continues to discharge slightly. Antrum shows no signs of filling in.

Oct. 14. Condition of antrum same as at note of Sept. 27. About one-quarter of posterior half of M. E. seen, red and granular. Bridge granular, and slight discharge from M. E.

Oct. 14. Discharged relieved to O. P. D.

August 5, 1912.						October 14, 1912.					
R					L	R					L
18					1	18					1
—		W			—	—		W			—
25					25	25					25
—		V			—	—		V			—
21		AC		25″	0?	30		AC		25″	15
—	F 512 V. S.	—	—	—	—	—	F 512 V. S.	—	—	—	—
13		BC		12″	13	26		BC		12″	25
	WEBER >	F 256 V. S.					WEBER	F 256 V. S.			
N		LIMITS GALTON			N	N		LIMITS GALTON			N
80 V. S.		LOW			V. S. 128	80 V. S.		LOW			V. S. 256

Vol. 34. page 281. Elizabeth Taylor, age 21. Birthplace, Mass. Residence, 22 Usher Road, West Medford. August 6, 1912.

Diagnosis: A. S. o. m. s. ch. Exostosis of canal.

Operation: Heath Radical Mastoid.

History: A. S. patient has had pain and discharge from A. S. for past 17 years, following scarlet fever. Has had treatment frequently since, beginning with discharge. Since August 4th she has had occasional pain. P. E. negative. Urine negative.

Examination: A. S. About 1 cm. from beginning of external auditory canal, canal closed entirely by a diaphragm of fibrous tissue at the superior part of which over an area the size of a large pin head there is crusting. Through this area probe can be passed into M. E.

Aug. 7. Operation, Ether. Mr. Heath. Usual Heath incision, etc., but in this case the bony exostosis was removed together with posterior canal wall. No ossicles found in M. E.

Aug. 8. Sutures removed and dressing reapplied.

Aug. 12. Posterior wound healed. Dressed every day and tube changed.

Aug. 16. Up and about. Small area of granulations coming from tympanic cavity.

Aug. 21. Antrum well filled in with granulations. Considerable amount of granulations over bridge and at posterior part of M. T. Incision in helix well healed.

Aug. 26. Tube has been shortened. Discharge less but still copious. Zinc sulphate, 10% to grans.

Sept. 2. Tube omitted. Dressed daily. Zinc sulph. to M. E. Scharlach Red to cavity.

Sept. 6. For past two days patient has complained of pain over original incision, which is tender to pressure. Considerable redness and swelling at lower part. Incision reopened and about one-half dram of pus expressed. Wicked. Hot sulpho-naphthol poultices. Cavity doing nicely and considerable part now epidermatized anteriorly.

Sept. 12. Infection mostly quieted down, but still a little discharge of pus from wound.

Sept. 19. Small area in lower part of incision which is not closed. M. E. closed from view by area of granulations. Up to beginning of old hyperostosis cavity is well epidematized.

Sept. 27. One-half inch from lower end of incision small fistula one-half inch in length. No bone felt and apparently does not connect with canal. Canal narrow in region of old hyperostosis beyond which a mass of granulations hide M. E. Posteriorly and superiorly probe strikes bare bone. Ag No_3 bead continued to granulations.

Oct. 5. Granulations in region of M. E. which repeated application of Ag No_3 bead does not keep down. Considerable amount of purulent discharge continues. Posterior sinus now healed.

Oct. 6. Region of M. E. filled with granulations except for small opening the size of pin head. This opening anteriorly reaches into region of M. E. Slight discharge continues from M. E.

Oct. 7. Discharged relieved to O. P. D.

August 6, 1912.

R				L
18				1
—		W		—
25				4
—		V		—
				40
14		AC	25″	0
—	F 512 V. S.	— —	—	—
10		BC	12″	3
	WEBER	F 256 V. S.		
N		LIMITS GALTON		N
80 V. S.		LOW	V. S. 2048	

October 7, 1912.

R				L
18				1
—		W		—
25				12
—		V		—
				40
15		AC	25″	0
—	F 512 V. S.	— —	—	—
20		BC	12″	0
	WEBER <	F 256 V. S.		
N		LIMITS GALTON		N
80 V. S.		LOW	V. S. 2048	

Vol. 35. Page 103. Name, Elizabeth Taylor. Age, 21. Birthplace, Massachusetts. Residence, 22 Maher Road, West Medford, Mass. Occupation, nurse. Nov. 12, 1912. (Re-entry.)

Diagnosis: A. S. o. m. s. ch. with recurrent mastoiditis.

Operation: Radical Mastoid. Skin graft (primary) Dr. Jack.

History: A. S. Heath operation by Mr. Heath, August 7, 1912. Discharge 17 years previous to operation continuing to present. Operated on six years ago by Dr. Plummer. Since operation has had a continual M. E. discharge and a gradually progressive narrowing of canal.

Examination: Canal is almost entirely obstructed with newly formed tissue. One small opening is seen, through which pus, of a muco-purulent character oozes. Slight tenderness over upper mastoid.

Nov. 20. *Operation.* Ether. Dr. Jack. Usual mastoid incision. Periosteum retracted. Cortex exposed and removed. Mastoid curetted out. Part of posterior wall was previously removed by Mr. Heath. Removal down to facial ridge completed. Small sinus leading to the dura over tegmen antic found. Antrum found and curetted. Ossicles removed.

Primary skin graft taken from right thigh, cut dry. Applied in mastoid cavity and sucked into middle ear. Held in place by a number of cotton pledgets. Wound packed lightly with iodoform tape and closed by four s. w. g. sutures. Thigh wound covered with cargile membrane and gauze.

Patient stopped breathing while coming out of ether, but recovered slowly.

Nov. 25. Stitches removed from posterior wound, also iodoform tape from axilla. Heart well compensated, and not enlarged pathologically. Urine negative.

Examination: A. S. shows well epidermatized radical cavity. Old scar over mastoid which is slightly reddened and swollen and tender in upper part.

Feb. 19. *Operation.* Ether. Dr. Jack. Incision in old scar. Old cavity exposed and well epidermatized in lower part. Cavity leading to dura filled with soft granulations. Dura normal. Grans cleaned out. Wound closed entirely with four s. w. g sutures. Iodoform gauze lightly packed into affected area through the canal. Dry dressing and bandage. Good ether recovery.

Feb. 23. Small granulation in bowl cauterized. Cavity looks well otherwise. Posterior wound doing well. Patient up.

Feb. 26. Doing well. M. E. still discharges, and granulations appear as above.

Feb. 29. Less discharge. Not many grans seen now.

Mar. 1. Discharged relieved to Dr. Jack.

PART II.

Abstracts of the cases. The abstracts are grouped according to classes, and are not given in chronological order. The following are the abstracts.

Case 1. August 12, 1912. Margaret Donovan. White. Age 6. American. Service of Dr. Plummer.

Diagnosis: Otitis media suppurativa acuta. A. S. Otitis media suppurativa acuta. A. D. Recurrent mastoiditis. Post aural abscess. A. D.

History: The patient was admitted to this hospital on Jan. 19, 1912, (i. e. eight months ago), suffering from otitis media suppurativa acuta and mastoiditis and post aural abscess A. D. A mastoid operation was done by Dr. Lothrop, the senior house surgeon, in which the abscess was incised and drained. The cortex was found to be perforated near the canal. The cortex was removed and all the soft cells curetted out. The antrum was found and curetted. The sinus was exposed but not opened. The patient was discharged on Feb. 1, 1912. The right ear was well until last Monday (one week ago), when the pain commenced and has lasted to date. There has been considerable discharge for the past three days. Mastoid tenderness has existed for four days, and swelling for three days.

Physicial Examination: Negative.

Examination: On the right mastoid there is a scar of the previous operation. There is marked post aural redness, bulging and fluctuation. A small round perforation exists in the lower posterior part of the drum. Through this comes a profuse purulent discharge. The drum of the left ear is red and bulging and no landmarks are seen. Under primary ether the left drum was incised.

Aug. 13. Operation. Heath conservative mastoid. Operator, Mr. Heath. Usual Heath incision. Pericranial flap cut. About half an ounce of pus escaped from under the periosteum. A small pin head perforation was found in the reformed lateral wall of the mastoid. The further steps of the operation were carried out in the manner usually followed by Mr. Heath.

Oct. 7. (One week short of two months.) The middle ear is dry. Most of the drum can be seen and the antrum has been filled with granulations and its site is epidermatized.

April 26, 1913. (Condition today.) The mother says that the child left the hospital four months ago with the ear dry and that it has remained so. She notices no impairment in the girl's hearing.

Examination: Two scars are present behind the ear. One

is one-half c. m. behind the crease where the auricle joins the head and the other one-half c. m. in front of this, and on the auricle. The incision on the auricle is the one made by Mr. Heath. In order to avoid the scar of the first operation Mr. Heath's incision was made either a little forward of the crease where the auricle joins the head or it has retracted into this position. There is no deformity of the crus helicus. The beginning of the cartilaginous canal is narrowed slightly at the top, but this is so little that it does not interfere with examination in any way. Within the canal and posteriorly and externally there is a slight excavation in the body of the mastoid. The cavity of the antrum has been obliterated flush with the anulus. The operative field is fully and firmly epidermatized. The bridge between the site of the antrum and the drum stands out clearly. The ear is dry. The whole of the lower posterior quadrant and half of the upper posterior quadrant of the drum is occupied by a thin atrophic scar. Two m. m. in front of the handle of the malleus and parallel with it there is a thickened line of calcification about one m. m. wide and four m. m. long. The drum is not retracted. The drum of the other ear is slightly dull.

Hearing test:

April 26, 1913.

R					L
18					18
—		W			—
25					25
18		AC		25″	20
—	512 v. s.	—	—	—	—
15		BC		12″	10
		WEBER			
N		LIMITS GALTON			N
96	SURE	LOW V. S.		SURE	96
64	?			?	64

Comments on this case: This is the best result of the seven. It was a case of recurrent mastoiditis and recurrent post aural abscess. The case was first operated upon by the simple mastoid operation with posterior drainage and then by Mr. Heath with drainage only through the canal. The records do not show that the post aural abscess was at either time as large and foul as in the other case of mastoiditis and

post aural abscess operated upon by Mr. Heath with less success.

Case 2. August 2, 1912. Joseph Weinstein. Single. Age 21. Russian. Service of Dr. Plummer.

Diagnosis: Otitis media suppurative acuta. A. D. Mastoiditis.

History: The patient has had a head cold for three weeks. With this he had pain in the right ear for one day. The drum was opened by his local physician, and the middle ear has discharged since. The pain stopped until two days ago, when it returned severely at intervals. There has been occasional headache and some mastoid tenderness.

Physical examination: Negative.

Examination: The mastoid shows considerable tenderness over the antrum and the tip. A profuse purulent discharge comes from the middle ear from a large irregular perforation in the posterior lower quadrant. There is a moderate amount of oedema of the superior canal wall.

Aug. 3. Operation. Heath conservative mastoid. Operator, Mr. Heath. Usual Heath incision, etc. On the seventh day after the operation the temperature rose to 105° and remained above normal for a few days. No cause was found. About one month after the operation, the antrum was one-half obliterated by granulations. For about two months the end of the posterior flap and the edge of the superior canal wall where it was incised in the making of the flap projected into the canal and interfered somewhat with the after treatment. At this time a note was made that the flap had gone back into place and that the incision through the crus helicis was healed without deformity and that the site of the antrum was represented by a slight dimple the cavity of which was well epidermatized.

Sep. 25. The patient has adenitis on the right side of the neck. This was treated with hot sulpho-napthol poultices. On the fifth day the right tonsil was removed. The patient did poorly under ether and the left tonsil was not touched. No point of infection was found.

Oct. 21. The adenitis is well, but the middle ear is still slightly moist. Two-thirds of the area of the drum can be seen. There is a small perforation in the lower posterior quadrant. The antrum is well epidermatized. The patient was discharged today to the Out-Patient Department.

April 26, 1913. Condition today, the patient did not report for examination.

Comment on this case: The antrum was obliterated and epidermatized at the end of two months. Insufficient drainage through the canal was the probable explanation of the temperature for a short time a week after the operation and for the infection of the neck later. The ear was not dry at the end of two months. A test made a week after leaving the hospital, showed no loss of hearing.

Case 3. July 31, 1912. Frank Sapanari. White. Age 30. Italian laborer. Service of Dr. Plummer.

Diagnosis: Otitis media suppurativa acuta. A. D. Mastoiditis.

History: Two months ago the patient had pain below the right auricle. It soon went away. One month later the ear felt blocked. The patient came to the Out-Patient Department and had cerumen syringed out of both ears. At that time he had otitis externa diffusa on the right ear, and on July 2nd, 1912, the canal was incised. Six days ago the ear began to discharge and a day later the mastoid became tender and has remained so since.

Physical examination: Negative.

Examination: The external canal of the right ear is filled with a polyp. There is a purulent discharge from the middle ear. No part of the drum can be seen. Marked tenderness is present over the antrum and the tip of the mastoid and at the site of the emissary vein.

Aug. 4. The polyp was removed from the right ear in order to give better drainage.

Aug. 8. The patient was examined with the naso-pharyngoscope by Dr. Emerson. The examination showed a sharp spur on the right side of the septum, and the spur was in contact with the turbinates. The posterior ends of the inferior

turbinates were large. The vault was congested, especially on the right.

Aug. 9. Operation. Heath conservative mastoid. Operator, Mr. Heath. Usual Heath incision, etc.

Aug. 28. (Nineteen days after the operation). The upper part of the skin incision has been healed for some time; the lower part has just healed. The incision through the crus of the helix has healed with gaping. There is a slight discharge round the rubber tube in the canal. There is a small tag at the middle of the superior wall. Inflation by the Valsalva method blows clear mucous through a perforation near the edge of the bridge. Zinc sulph. applied.

Sep. 27. The canal is narrowed a little externally. Superiorly a ridge projects from the canal wall. There is an opening into the antrum 2 mm. in diameter. From this comes a pulsating discharge. The drum is hard to distinguish. There has been a profuse purulent discharge from the middle ear until the last three days. Drops of boracic acid and alcohol solution were ordered to be put into the ear twice daily. The granulations were touched with a bead of nitrate of silver.

Oct. 10. (One week less than two months after the operation). For the past few days the discharge from the middle ear has been less than before. The antrum, however, has not filled in.

Hearing test at entrance. Aug. 3, 1912. No record of hearing test at discharge.

August 3, 1912.

R				L
1/6				18
—		W		—
25				25
5		AC	25″	10
—	512 V. S.	—	—	—
11		BC	12″	15
		WEBER <		
N		LIMITS GALTON		N
128		LOW		80

April 26, 1913. (Condition today). This case did not report for a final examination. He came to the Out-Patient De-

partment very irregularly. The last note on the case was made December 16, 1912, four months after the operation. The note reads: The discharge from the ear stopped one month ago or three months after the operation. The cartilaginous canal is slightly narrowed. There is no deformity of the crus of the helix. The operative excavation in the mastoid is represented by a slight bowl. The antrum is fully granulated and epidermatized. Only the lower third of the drum can be seen. The ear is dry.

No final hearing test is recorded.

Comments on this case: At the end of a month the antrum had granulated in one-half, but at the end of the second month obliteration of the antrum had not yet been accomplished. However, judging from the last note, (of the record), the antrum filled and became epidermatized somewhere in the third month, and the discharge stopped.

Case 4. August 14, 1912. Louis Kaplan. Age 2. American. Service of Dr. Plummer.

Diagnosis: Otitis media suppurative acuta. A. S. Mastoiditis. Post aural abscess.

History: The child has had a discharge from the left ear for the past ten days. Swelling developed over the mastoid region six days ago.

Physical examination: Negative.

Examination: On the left side there is post aural redness, oedema and tenderness. Palpation shows a well localized post aural abscess. The drum is red and bulges posteriorly. No perforation is seen. There is considerable purulent discharge in the canal.

Aug. 15. Operation.Heath conservative mastoid. Operator, Mr. Heath. Usual Heath incision, etc. A large and very foul post aural abscess was opened. This burrowed upward under the temporal muscles. Owing to the extent of the abscess it seemed to one of the writers (Dr. Mosher), that drainage through the canal only, as Mr. Heath proposed to do, might not be sufficient. At the close of the operation he asked Mr. Heath about this, and Mr. Heath answered that he thought it would be sufficient.

This case did not do well. The superior canal wall hung down and the posterior flap sprung forward from the bowl of the mastoid excavation. Both the posterior flap and the superior canal wall became boggy and thick and practically blocked off the middle ear. The incision through the crus helicis spread widely so that it became necessary after a week to withdraw the rubber drainage tube from the canal and to substitute a gauze wick. The skin incision healed, but on the tenth day broke open at the lower part. The ear continued to discharge both through the canal and through the post auricular opening so that about a week short of four months after the operation. Dr. Jack reoperated the case.

Dec. 5, 1912. Operation. Radical mastoid. Operator, Dr. Jack. The incision was made close to the auricle in order to repeat the original incision. The remaining steps of the operation were those of the familiar radical mastoid exenteration. A small skin graft was placed in the cavity. The incision for the radical did not unite and the dressings were done from the post-auricular opening.

Since leaving the hospital the child has been brought to the hospital once a week for treatment of the radical cavity.

April 26, 1913. (Condition today.) There is a large post auricular wound. The bowl of the radical mastoid cavity is packed with plain gauze, and a small iodoform wick fills the middle ear. On removing the wicks the bowl of the radical cavity is found to be well epidermatized. The antrum and middle ear, however, are not epidermatized, and small, flat granulations are present in the antrum. The middle ear is narrowed from above downward, so that one-third of its cavity has been lost.

No hearing test.

Comment: The natural comment on this case is that the drainage of the post aural abscess was insufficient and being insufficient the case went on to a radical mastoid operation whereas in most of such cases the simple mastoid operation and ample posterior drainage would have been all that was necessary. The radical mastoid operation produces a vastly

greater loss of hearing than the simple operation. It should be borne in mind, however, that cases of this kind at times do badly and require more than one operative interference.

Case 5. July 19, 1912. Russell Noyes. Age 12. American. Service of Dr. Hammond.

Diagnosis: Otitis media suppurativa chronica. A. S.

History: About once a year for the past six years, the patient has had at times a discharge from the left ear. This was accompanied by deafness.

Physical examination: The left ear shows a large perforation which involves the posterior half of the drum. The incudostapedial joint is exposed and is covered with granulations. The promontory is similarly covered. There is considerable purulent discharge. The right drum is atrophic.

July 29. Under local treatment the ear has shown no signs of improvement.

Aug. 12. Operation. Heath conservative mastoid. Operator, Mr. Heath.

Oct. 19. The house record reads as follows: "The antrum is granulated except for a cavity 4 mm. in diameter and 4 mm. deep. There is a very slight moisture in the middle ear. The anterior half of the drum is covered with granulations."

Dec. 28. (Four months after operation). The last house record reads: "Considerable discharge. Slight moisture in the antrum, but antrum well epidermatized."

April 26, 1913. (Condition today). Since leaving the hospital the mother says that the boy's ear was dry until two months ago. It is probable, however, that the ear has never been absolutely dry. The boy has been coming to the Out-Patient Department once a week for treatment, and drops of the usual boracic acid and alcohol solution have been used in the ear most of this time. According to the mother's story two months ago, after a shower bath at school, the ear began to run and discharged for two weeks. After the exacerbation attributed to the shower bath, the discharge was not noticed again until one week ago. The mother thinks that the operated ear falls away from the head a little more than the other ear does.

Examination: Both ears are large. There is a very slight lop ear on the operated side. The scar of the incision is in the crease where the auricle joins the head. There is a slight deformity of the crus helicis, i. e., the posterior half of the crus is a little smaller than the front half and where the two join, there is a very slight angular deficiency at the top and bottom of the scar. The cartilaginous canal is slightly narrowed especially superiorly, for 1 cm. inward. A good sized speculum can be made to pass the narrowing easily. Two drops of pus were wiped out of the canal. Where the posterior wall of the canal was taken down at the operation and an excavation made in the mastoid, no cavity now remains.

One-third of the antral cavity is obliterated, epidermatized and dry. On the superior wall of the canal half way to the drum a good sized polyp hangs downward. At the root of this the probe detects a spot of necrosis. The bridge between the antrum and the drum stands out clearly. The drum has no landmarks, and is thickened and covered with epithelial débris. In the posterior inferior quadrant, there is a two mm. perforation. Through this when the patient practices inflation by the Valsalva method, soft granulations and pus are blown. Behind the perforation and on the edge of the anulus there are a few soft granulations.

December 28, 1912.				April 26, 1913.				
R			L	R				L
18			15	18				5
—	W		—	—	W			—
25			25	25				25
36	AC	25″	17	22	AC	25″		18
—	512 v. s. —— — ——		—	—	512 v. s. —— — ——			—
21	BC	12″	14	15	BC	12″		13
	WEBER <				WEBER <			
N	LIMITS GALTON		N	N	LIMITS GALTON			N
96	LOW LIMIT		96	64	LOW LIMIT		96 i. e. G.	

Comment: Sometime between the third and the fourth month the antrum granulated in as much as it was going to and become epidermatized. The discharge lessened during the treatment, but the ear has never been seen dry at the hospital. If this case had been operated by either of the writers of this report a radical mastoid operation would have been performed. Eight months, of course, is a long time to

have a radical cavity remain moist, but many of us have had such cases. The middle ear is still involved and what the final outcome of the case will be, is not yet clear. Since leaving the hospital there has been a marked loss of hearing, but the hearing is better than one would expect from the appearance of the ear. As the antrum has been partly obliterated and is dry, it would seem that the ossicles were responsible for the continuance of the trouble.

Case 6. August 5, 1912. Daniel Murphy. White. Age 13. American. Service of Dr. Plummer.

Diagnosis: Effectus otitis media suppurativa chronica. A. S.

History: Acute exacerbation.

Three weeks ago the present attack started. Mastoid tenderness developed this morning, but there was no swelling. The patient gives a history of a similar attack one year ago.

Physical examination: Negative.

Examination: There is a profuse purulent discharge from the middle ear on the left.The drum is red but not bulging, and there is a small perforation in the lower posterior quadrant. The patient cannot blow air through the perforation. Moderate tenderness is present over the antrum of the mastoid but not at the tip. Neither redness nor oedema is present.

Aug. 9. The patient has remained the same since admission. Mr. Heath saw him several times and advised operation. The tenderness is less. A profuse discharge continues from the middle ear. Transillumination shows a slightly light antrum and a dark tip.

Aug. 10. Operation. Heath conservative mastoid. Operator, Mr. Heath. Usual Heath incision and operation.

Aug. 28. The following note was made in the house records on this date, and as it is in some respects a characteristic note, it is given entire. "The upper third of the incision is granulating. The lower third of the incision is granulatng also. The incision through the crus of the helix is healed without deformity. There is a moderate amount

of pus about the rubber drainage tube which holds the canal open. The wick which passes through this is well saturated with pus. The inner end of the canal flap projects slightly. The anterior border of the canal flap and the roof of the canal project slightly. The antrum shows but a few granulations about the edges. The bridge is covered with granulations except superiorly. On the anterior surface of the bridge there is a small rim of granulations which obscure the upper third of the drum. Inflation by the Valsalva method is successful and muco-purulent discharge is blown from the middle ear. The rise of temperature which occurred two days ago has subsided. A gauze wick is placed in the antrum and another carried to the drum. The white blood count is 20,000." The typical part of this note consists in what is said about the granulations from the bridge and the displacements of the segments of the canal.

Oct. 4. The middle ear continues to discharge slightly. The antrum shows no signs of filling in.

Oct. 14. This note was made two months after the operation when the patient was discharged to the Out-Patient Department. The condition of the antrum is the same as in the last note of Sept. 27, i. e., no tag or flap is present in the region of the antrum. The cavity of the antrum is represented by a hole 4 m. m. in diameter into which a probe enters. Today, October 14, "About one-fourth of the posterior half of the drum can be seen. The drum is red and granular. The bridge is granular and there is a slight discharge from the middle ear."

April 26, 1913.
Hearing Test at Entrance.

R			L
18			1
—	W		—
25			25
2	AC	25″	0?
—	— —	—	—
13	BC	12″	13
	> WEBER		
N	LIMITS GALTON		N
80	LOW LIMIT		128

April 26, 1913.
Final Hearing Test Seven Months After Operation.

R			L
18			1
—	W		—
25			25
33	AC	25″	5
—	— —	—	—
18	BC	12″	18
	WEBER <		
N	LIMITS GALTON		N
64	LOW LIMIT		? 96-G.

512 V. S.

April 26, 1913. (Condition today.) The mother says that the cotton in the ear is always moist, and that the ear has never remained dry for more than one week since the operation.

Examination: The scar of the incision is in the crease where the auricle joins the head. There is a slight deformity of the crus helicis with narrowing of the canal especially superiorly for 1 cm. inward. There is a small amount of secretion in the canal. There is no bowl in the mastoid corresponding to the operative excavation. The antrum apparently is not filled in at all and the cavity of the antrum is moist. A cresentic fold of mucous membrane springs from the bridge and is parallel with it. This fold at the top measures about 1 mm. from before backward. There is a good sized perforation in the posterior, inferior quadrant of the drum, while the remainder of the drum is thickened, boggy and without landmarks. Inflation by the Valsalva method is not successful, either through the drum, or through the aditus and antrum. A probe introduced into the antrum apparently enters the aditus.

Comments on this case: After seven months the antrum has not filled in. The middle ear is still moist and there is a great loss of hearing.

Case 7. August 6, 1912. Elizabeth Taylor. Age 21. American. Nurse. Service of Dr. Plummer.

Diagnosis: Otitis media suppurativa chronica. A. S. Exostosis of canal.

History: The patient has had pain and discharge from the left ear for the past seventeen years, following scarlet fever. Since the beginning of the discharge she has had much treatment. Since Aug. 4th of this year, she has had occasional pain.

Physical examination. Negative.

Examination: About 1 cm. from the beginning of the external auditory canal, the canal is closed entirely by a diaphragm of fibrous tissue at the superior part of which over an area the size of a large pin head there is crusting. Through this area a probe can be passed into the middle ear.

Aug. 7. Operation. Heath radical mastoid. Operator, Mr. Heath. Usual Heath incision, etc. The exostosis was removed together with the posterior canal wall, and thus a new bony canal was formed. No ossicles were found in the middle ear. (The presence of the exostosis made the finding of landmarks and the performance of the operation very difficult. Comp's.)

This case did not do well and was finally reoperated three months later by Dr. Jack.

Examination: The canal is almost entirely obstructed by newly formed tissue. A small opening is present through which pus of a muco-purulent character oozes. There is a slight tenderness over the upper part of the mastoid.

Nov. 20. Operation. Radical mastoid operation. Operator, Dr. Jack. Usual mastoid incision. Periosteum retracted, and the cortex exposed and removed. Mastoid cells curetted out. As only a part of the posterior canal wall had been removed by Mr. Heath, the removal of the wall was completed down to the facial ridge. A small sinus was found, which led to the dura over the roof of the attic of the middle ear. The lateral sinus was far forward. The antrum had not been opened in the operation of Mr. Heath. The antrum was found by Dr. Jack and curetted. The ossicles which were not found in the first operation, were located and removed. A primary skin graft was placed in the operative cavity.

Three months later, the scar over the mastoid became reddened in the upper part and swollen and tender.

Feb. 19, 1912. Operation. Reopening and curetting of radical cavity. Operator, Dr. Jack. Incision in old scar. The old cavity was exposed and found to be well epidermatized in the lower part. The cavity over the middle ear which had been taken by Mr. Heath for the antrum, was filled with soft granulations. The dura was normal. The granulations were cleaned out. The wound was closed entirely, and iodoform gauze packed lightly into the curetted area through the canal.

Feb. 23. A small granulation in the bowl of the mastoid cavity curetted. The cavity looks well otherwise. The posterior wound is doing well.

Feb. 26. The middle ear still discharges. Granulations in the bowl.

Feb. 29. The discharge has lessened and there are not many granulations.

Mar. 1. The patient was discharged relieved to Dr. Jack. When last seen by Dr. Jack, the radical cavity was epidermatized and dry.

April 26, 1913. (Condition today.) The patient was out of town and answered by letter that she would report for examination when she returned.

The comments on this difficult case which the compilers would make are as follows: (Before this case came to the hospital, it was operated by Dr. Plummer and the exostosis removed. When Mr. Heath operated, the exostosis had returned.) Mr. Heath's incision and partial removal of the posterior wall and the mastoid cells caused him to miss the antrum and to open the middle fossa, and to consider the operative cavity thus made as the antrum. A complete tympano-mastoid exenteration became necessary at the hands of Dr. Jack. The primary skin graft put in by the second operator was in great part successful. The cavity above the attic of the middle ear refilled with granulations, did not drain and infected the upper portion of the scar. This necessitated the reopening of the radical cavity and a second curetting.

Summary of Results.

The seven cases operated upon by Mr. Heath fall into the following groups: Two cases of otitis media suppurative with mastoiditis; two cases of otitis media suppurativa acuta with mastoiditis and post aural abscess (one of these cases was recurrent); three cases of otitis media suppurativa chronica. Two of the seven cases required re-operation, i. e., one case of mastoiditis with post aural abscess which came to a radical operation, and one chronic case, the one that had the recurrent exostosis of the canal. This also came to a radical operation.

Of the acute cases two are known to be dry and to have the antrum filled in and epidermatized and to have whole

drums. The shortest time in which the middle ear became dry was seven weeks The second acute case had the middle ear dry, and the antrum obliterated and epidermatized somewhere in the third month. The third acute case had the antrum epidermatized and dry in two months and a half, but the middle ear was still moist.

Two of the three chronic cases are still discharging after seven months. In the first of these the antrum is one-third obliterated and epidermatized; but in the second the antrum has not filled in at all.

Of the two re-operated cases, the first one, the two and a half year old child with post aural abscess which ended up with a radical, is two-thirds healed. The second case, the one with the recurrent exostosis which required a full radical, is well.

The results as to hearing in the seven cases are as follows: In the first of the acute cases there has been no impairment; in the second the hearing was not impaired when the patient was discharged from the hospital; in the third, the last note says that the antrum was granulated flush with the anulus and epidermatized and that the drum was normal. In this third case, although there is no record of the fact, it is fair to presume that little if any impairment of hearing has followed the operation. In the fourth and the last of the acute cases a radical operation was necessary so that a severe loss of hearing has resulted. As the patient is only two and a half years old no test has been made. Of the three chronic cases one shows a slight loss of hearing since operation, and one a large loss; whereas the third case which was practically deaf in the diseased ear, probably shows but little loss. This case did not report for final examination. (See above.) A test made in the middle of her hospital stay and just before the radical operation, showed but a slight advance in the deafness. To sum up the hearing results, it can be said that in three of the four acute cases there was no loss of hearing. One of these cases required a radical operation and so sustained a severe loss of hearing. In the chronic cases one had a slight loss, one a severe loss

and in the third, the ear was practically deaf and there has been but slight change for the worse.

The subject of the paper, as stated in the beginning, is "A Report of the Results of Seven Cases Operated upon by Mr. Heath." With the preceding paragraph, therefore, this report has reached its logical end. There are other topics, however, which are vitally connected with the Heath operation that have not been touched upon. There is great risk if any of these are now brought up of spoiling the symmetry of the paper and of starting an avalanche of discussion. Notwithstanding this, the writers wish to continue for a few more paragraphs and to make some observations on the after course of the operated cases. They hope in so doing that they do not offend. First of all it should be borne in mind in considering these seven cases that they are relatively too few in number to draw hard and fast conclusions from them. Moreover, when a surgeon operates away from home and leaves the after treatment of his cases to others the results are sure to be less favorable than they would have been had he finished the cases himself. A letter recently received from Mr. Heath called attention to this fact. Every allowance should, therefore, be made. On the other hand, however, if Mr. Heath's operation is to be done by anyone except himself, the after treatment must not be so difficult that men of average ability cannot carry it out. You may put me in that class if you like, but I will not permit my superior, Dr. Plummer, to be spoken of in that way.

So much for that point. Now for the others. The scar left by the skin incision of Mr. Heath, coming as it does in the crease where the auricle joins the head is practically invisible, and is the best looking scar known to the writers. This is all that can be said in its favor, because by making the incision so far forward the surface of the mastoid is not sufficiently uncovered to reveal all possible foci of disease. The tip of the mastoid and the lateral sinus are not easily dealt with through such an incision. When Mr. Heath was questioned about this, he replied that he always stood ready to make a supplementary horizontal incision. He did

not find it necessary, however, to do this in any of the seven cases, in spite of the fact that in one of them he missed the antrum, and unknowingly and fortunately missed a forward lying sinus. Not only would the additional incision have helped greatly in this very hard operation, but if he had attacked the mastoid through the outer wall in the usual way, instead of through posterior canal or all of the canal, as is his custom, the case would have been infinitely easier and probably more successful. Mr. Heath's incision through the crus of the helix gives a temporary widening of the canal which is very useful. It heals with little if any deformity.

For the first week after Mr. Heath's operation it is a surgical pleasure to look through the widely open canal and to see in the field behind and above, the round opening of the antrum, and in front the whole of the drum, while between the antrum and the drum the glistening bone of the bridge stands out sharply. The pleasant impression produced by this is increased when one looks behind the auricle and sees the almost invisible scar. One is impressed agreeably also, by the fact that there are no wicks to be put in and taken out through a gaping posterior wound. Everything can be done through the canal. A bit later, when in spite of all care, granulations begin to spring from the bridge the picture changes to a less pleasing one. Granulations bud from the bridge and from the posterior and the superior part of the anulus, and soon obscure a large part of the drum and dam back the discharge. At the same time, in some cases, the superior wall of the canal sags and the flap made from the posterior wall breaks from its moorings and projects forward into the lumen of the canal, obscuring the view and hampering the dressings. The segments of the canal wall finally go back into place and the end results show that the extensive upset of the canal which occurs at the operation and continues for a time afterwards produces only a slight and insignificant narrowing. The most serious drawback in the convalescence of the cases is caused by the granulations which spring from the bridge and anulus, because they block off the drum, wholly or in part. In three

or four of the acute cases the antrum filled flush with the anulus, and epidermatized and the ear became dry. But in none of the three was the time necessary to accomplish these results as short as is often the case when such cases are operated upon in the usual way. None of the chronic cases in our opinion did as well as they would have done had they been operated by the full radical mastoid operation with cleaning out of the middle ear. The serious defect of the Heath operation is that it attempts to deal with the mastoid process from an awkward angle, and through a restricted space, namely the posterior canal wall. The operation has something about it of the nature of a stunt. It can be done and done successfully. The vital defect of the operation and the thing that is continually liable to happen in this method is insufficient exposure of the mastoid surface, and incomplete removal of pathological conditions within it. Finally, and this observation brings up another point in connection with Mr. Heath's operating, we are under great obligations to him for reviving the question of operating early, both in acute and chronic cases, in order to preserve as much of the hearing as possible. Of late this reason for operating has fallen somewhat into the background. Mr. Heath has brought it vigorously to the foreground again. The writers of this paper wish to acknowledge the stimulating effect of Mr. Heath's visit. They wish to praise his mechanical dexterity, and his generous personality, and to express their appreciation of the force of a man who could make the otological world stand, we cannot say stand and deliver, for that is not true,—who could make the otological world stand and re-consider.

Discussion.

Dr. Frederick L. Jack, of Boston, Mass., thought it only fair to Mr. Heath to say that in one of the cases upon which he operated in Boston there was a very large exostosis which hindered him in doing his own operation. The exostosis was very large and there was considerable dura exposed at the time. The cavity filled with granulations. The speaker

performed the usual radical operation with primary graft, and got good healing. But as Dr. Mosher said, there were one or two cases in which healing took place rapidly, and the results were as good as they would have been from the ordinary radical operation.

THE USE OF VACCINES AFTER THE MASTOID OPERATION.

By WILLIAM C. BRAISLIN, M. D., Brooklyn, N. Y.

I have employed vaccines in six cases, subsequent to the mastoid operation, out of a total of thirty-five mastoid operations performed during the past winter.

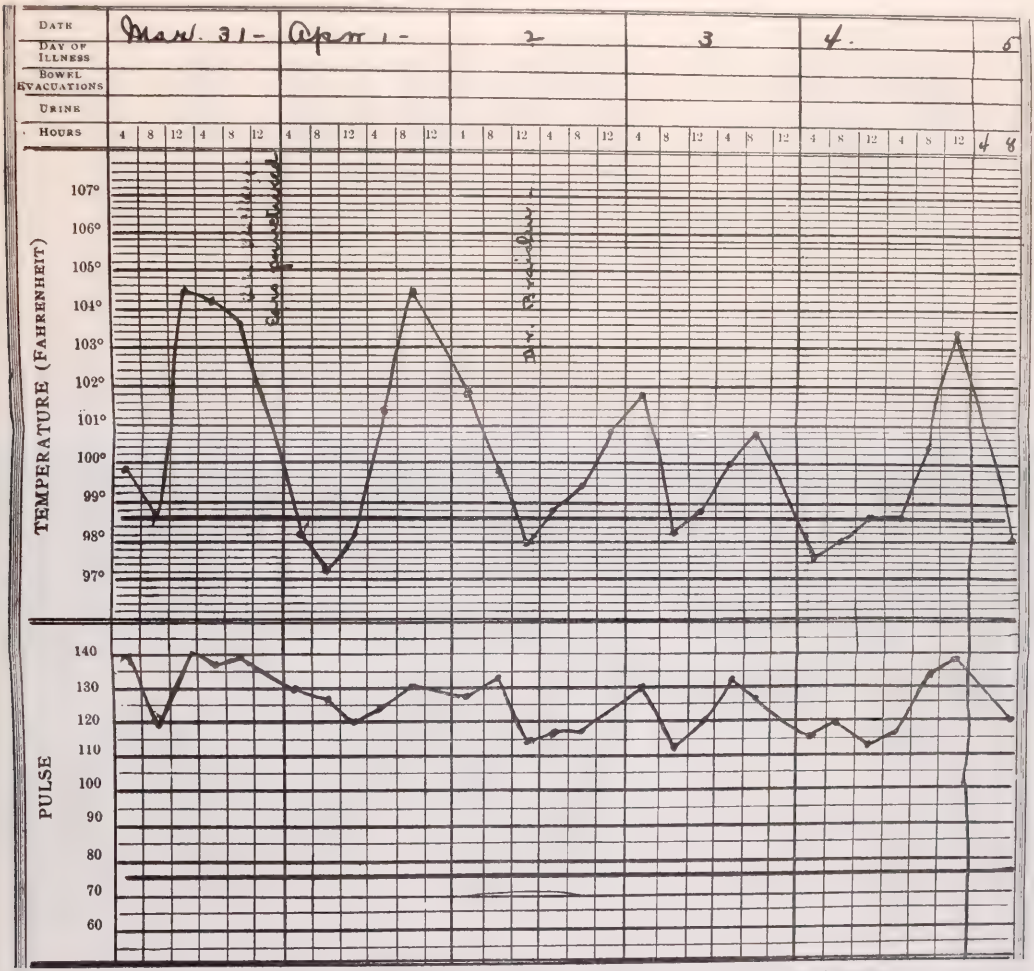
In the ordinary, smoothly convalescing cases it has not been used.

It was used in cases presenting the following complications:

1. Extensive infiltration of the glands of the neck following an operation for streptococcic mastoiditis and cervical abscess.
2. Indeterminate or obscure causes of elevated temperature (the lateral sinus excluded from involvement).
3. Delay in granulation and healing processes in mastoid wound.
4. Pain in limbs, joints and back, without special elevation of temperature, after an operation for streptococcic mastoiditis.
5. In a case of post-operative pneumonia.
6. In a case of infection of the labyrinth.

Case I. Streptococcic infection of right mastoid in a little girl of two and a half years.

The mastoiditis was complicated in this case by a very extensive abscess on the corresponding side of the neck which was operated upon at the time of the mastoid operation and a drainage tube introduced into the depth of the wound. The cervical abscess was deeply seated and surrounded by enlarged lymphatic glands, one of which protruded into the right lateral and posterior wall of the pharynx. A culture taken at this time and submitted to Dr. Archibald Murray, showed the infection to be streptococcic. The child did well for



Pneumococcus infection of both mastoids at beginning of second week after eruption of measles

three days after the operation, but the following day the lymphatic glands of the neck on the opposite side became tense, markedly swollen and tender, especially at the angle of the jaw, and a distinct rise of temperature occurred. No involvement of the mastoid was discoverable on this (the left) side. The culture made at the time of the operation not being available for making an autogenous vaccine, we began at once the use of a stock (streptococcic) vaccine, a minim being given daily for four days, then on alternate days, for four days more. The glandular swelling had begun to decline within twenty-four hours of beginning treatment. The discharges of pus from the wound lessened rapidly likewise. The temperature became gradually normal, and there appeared to be a sensible increase in the rapidity of the healing of the mastoid wound. The neck wound healed within three weeks from the time of operation; the mastoid wound, at the end of the sixth week.

Case II. Pneumococcus infection of both mastoids, ten days after eruption of measles, in a girl three years old. Both drums were promptly incised. Cultures taken from both ears were reported by Dr. Murray to be pneumococcic. The discharges continued to be profuse from both ears, and the temperature continued high. The right mastoid which was tender to pressure was operated upon, (April 7) the sinus being exposed, but healthy. The antrum and cells contained much free pus. An autogenous vaccine from a culture removed from the antrum was started in course of preparation, at our request, by Dr. Murray. Much to my disappointment the temperature did not subside to normal, but continued nearly as high as before the operation, between 102° and 104° so that after excluding the lungs from any influence in the production of the temperature, the left mastoid was operated upon on the third day following the operation on the right. Flaky pus was found in the antrum. We now believed that the temperature would fall to normal, but in this we were again disappointed. Some few pus cells were found in the urine at this time, but not enough to indicate a pyelonephritis. The lungs remained clear; though mucus and some blood ap-

peared at times in the stools, indicating a condition of the colon which affected the temperature in some degree. Three days after the second operation, the autogenous vaccine was ready for use, the first culture having failed to grow with sufficient activity to provide a vaccine.

Some reaction followed the inoculation of the vaccine, the temperature even rising to 105° the second day after its use. From this point on, a rapid subsidence of the temperature took place. On April 24, a small skin abscess of the scalp which had newly developed, with a slight rise of temperature was incised. Both wounds are doing well.

Case III. A tall girl of fourteen years, large for her age, always rather delicate, and has grown very rapidly for the past two years. Her mastoiditis, of four days' duration, followed a severe attack of tonsilitis, accompanied by profuse discharge from the ear, extreme tenderness of mastoid and high temperature.

At operation (Jan. 10), an extensive oedema existed in the tissues over the mastoid. Blood-stained serum was encountered throughout the diplöetic mastoid. Free pus was found directly in contact with the lateral sinus which was exposed externally and anteriorly, for a length of three-quarters inch, but was apparently healthy. After the operation the temperature rose but once over 100° F., but the patient complained greatly of pain in her limbs and back. This continued to be the cause of much complaint, and on Jan. 14 one-half c. c. of an autogenous streptoccic vaccine, prepared by Dr. Murray was given containing 50 million bacteria to the c. c. The afternoon following the first dose the temperature rose to 101.2° with an aggravation of the pain in the limbs and back. The next day she was much more comfortable than before the inoculation, the pulse rate became slower and stronger by the second day, and on the third day the pain had left the limbs and she seemed brighter and more comfortable. Throughout her convalescence, which was rather prolonged, more or less tenderness of the joints persisted and a mild attack of chorea occurred about the time of the com-

plete healing of the mastoid wound. I am inclined to believe that these symptoms would have been less in degree had we persisted in the use of the vaccine.

Case IV. Harry Spencer, 9 months. A delicate-looking, wasted infant of mulatto (West Indian) parentage, was received at the Brooklyn Eye and Ear Hospital for operation. There was a tense swelling above and behind the right ear. A purulent discharge from the ear had existed for an indefinite period. Operation was performed Nov. 19th, 1912. On incision much pus was liberated, besides which, the swelling contained flaky fibrinous masses of a cheesy consistency, which were curetted away. A sinus in the bone led to the antrum. The malleus and incus, which were removed, appeared necrotic. A small area of healthy looking dura was exposed above and posteriorly. Two weeks later a swelling appeared on the opposite (left) mastoid region, and on Dec. 5th, this was also operated upon, this mastoid being operated upon by Dr. Bouvier, senior house surgeon of the hospital, who reported that thick curd-like flakes of pus similar to the condition found in the right had been likewise found in this (the left) side. Neither wound made the least progress toward healthy granulating or healing. Mercurial inunctions were prescribed but an irregular septic temperature developed and the child became still more emaciated. A polyvalent vaccine (Van Cott's) was administered every fourth day for a period of four weeks. The child died Feb. 6, 1913, having been in the hospital ten weeks. Dr. Murray performed an autopsy. Both mastoid wounds were clean, but almost as little healed as when operated upon. The brain was entirely normal and uninfected. The dura was exposed over three small areas, two on the right, one on the left side. Both lungs were extensively consolidated. A plurisy of a sero-fibrinous type was present, the fibrin being deposited in thick layers. A very large abscess was found in the middle of the right lung.

Case V. Radical mastoid for chronic suppuration: infection of the labyrinth.

A boy of 11 years was operated on Nov. 15, for chronic suppuration of the left ear, existent since infancy. A radical mastoid operation was performed. The incus was absent. Vomiting persisted for three days after operation. No nystagmus, tongue coated. Calomel was ordered after which the bowels moved and the nausea ceased. Two days later (21st) the temperature rose to 102° F. (mouth) and persistent vomiting recurred, especially on rising in bed. Vertigo as if falling forward (not toward the affected side) and severe headache. Slept but little. Next day more comfortable. Mind perfectly clear. Some stiffness of neck, temperature remitted, but again rose to 102° in the afternoon. Polyvalent vaccine one-half c. c. was administered every four days, the temperature continuing irregular for a period of three weeks, meanwhile assuming a flatter curve and a gradual cessation of all untoward symptoms. Ear entirely healed and dry at the end of nine weeks, but hearing *nil* in this ear.

Case VI. A man, 36 years of age, with marked mastoid tenderness, severe pain left ear, for six days profuse discharge from left ear, with a history of previous ear trouble. Operated Feb. 27, 1913, the culture from mastoid antrum was reported by Dr. Murray to be pneumococcus.

The next day he complained of severe pain in the right side of the chest, especially on deep breathing. Dr. Ritter, senior house surgeon, applied wide bands of adhesive plaster. The temperature on the next day, (Mar. 1), rose to 104.4°. No consolidation could be detected. On the next day, Dr. Frank L. Tucker examined the chest at my request and found a pneumonia area of consolidation in the right lung being clearly made out. On March 3rd, and for three days thereafter a stock pneumo-vaccine was administered daily, each dose representing 20 to 40,000,000 bacteria. On March 4th, the temperature came down to 99.4° with profuse perspiration and did not after rise above 101°. Recovery rapid and without further complications.

Discussion.

Dr. Stephen H. Lutz, of Brooklyn, N. Y., felt sure that the vaccines have a distinct field of usefulness in otology. Some misunderstanding had been caused by the indiscriminate use of vaccines without proper bacteriological examination. The use of stock vaccines, or those containing several varieties of organisms, did not appeal to him as being very scientific, but with proper bacteriological examination and the administration of the proper vaccine in a given case, he thought good results could be obtained. That had been his experience at the Brooklyn Eye and Ear Hospital.

Dr. S. MacCuen Smith, of Philadelphia, said the important question with reference to vaccines was whether an autogenous or a mixed vaccine be used. He cited a case in which he was asked to open a tonsillar abscess, with swelling in the neck. After he had opened the swelling in the neck one finger could be passed through the opening in the neck and one through the opening of the tonsillar abscess and they would meet. Notwithstanding these operations, the temperature continued to rise. Phylacogen had been recommended by another physician, who had brought some with him, but it was decided to wait until next morning before giving it. The next morning the patient's temperature was normal and remained so, nothing having been given. He cited this case to show what phenomenal results would have been attributed to this preparation if it had been employed.

Dr. Braislin, in closing the discussion, said he had used the vaccines for years with a skeptical spirit,—that is, as a last resort. He had latterly met with so much greater success, however, in these several cases, that he felt enthusiastic, and believed more and more good would be accomplished by this method of treatment as the products improved, particularly after operative measures had been resorted to, to remove the suppurative focus.

THE IMPLANTATION OF CARTILAGE FOR THE PREVENTION AND CORRECTION OF DEFORMITY OF THE AURICLE.

By HARRIS PEYTON MOSHER, M. D., Boston, Mass.

Fifteen days after a radical mastoid operation on the right ear the patient, a girl of sixteen, whose surgical misfortunes are the subject of this short communication, interrupted a previously normal convalescence with an attack of acute tonsillitis. In a few days this infected her well ear, and caused a violent middle ear suppuration. As if this was not affliction enough five days later the auricle of the right ear, that is, the ear on the side where the radical had been done, became red and tender. At first I thought that this was due to erysipelas, and certainly hoped that it was. Unfortunately it proved to be a septic chondritis. After ten days backing and filling on my part during which time an incision was made into the most prominent part of the swelling of the auricle and no pus was found, the patient was etherized again and an incision made parallel with the helix and nearly as long. This allowed the skin of the whole front of the auricle to be turned forward and the septic cavity to be curetted. No pus was found. Probably the reason for this was that it had drained into the canal through the incisions which were made for the auricular flap at the time of the mastoid operation. Two-thirds of the auricular cartilage had disappeared. Only in the lower part of the ear was there any left, and this was small in amount.

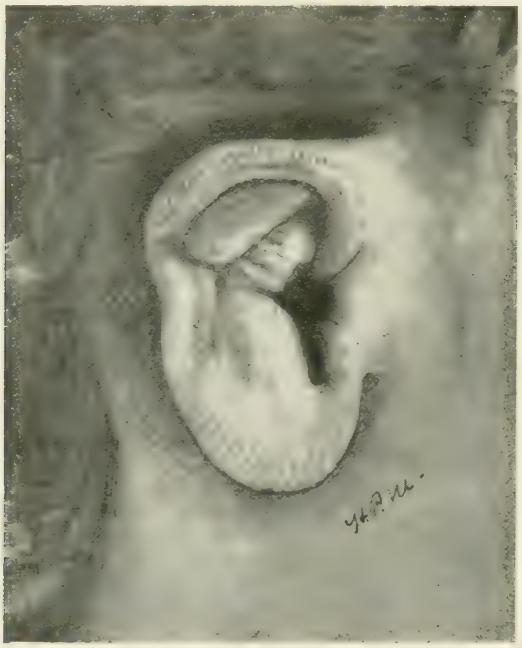
The wound was wicked and treated in the usual way for a septic cavity. After sixteen days the suppuration had stopped microscopically. From now on the only question was how much the auricle would shrink and what would be the extent of the resulting deformity. The large loss of cartilage meant the final shrinking of the ear into a shapeless mass. To stand idly and hopelessly by and watch the

process go on without making any attempt to check it was most unpleasant.

For five years, at least, I have been using freely transplanted septal and rib cartilage for the correction of deformities of the nose. I have employed cartilage also for the correction of sunken scars left by extensive Killian operations. With this experience in mind I decided to try what cartilage would do in this case. Accordingly, on the sixteenth day after the curetting of the auricular abscess the incision was in part reopened and the wound cavity again cleaned. Then a piece of cartilage was removed by submucous resection from the patient's nose and placed in the upper part of the auricle. A similar piece borrowed from the nose of another patient was put in the lower part. This had been kept in cold storage for twenty-four hours in anticipation of this use. The skin incision was sutured lightly and drained. Naturally first intention was not to be expected and was not obtained. The suppuration, however, was of slight severity and after a moderate period of wicking, ceased, and the wound closed. Twenty days later the cartilage was still in place; only a third had been absorbed. Encouraged by this result it was decided to try to paint the lily, as it were, and to put in more. Cartilage was obtained from two cases of submucous resection, and one piece was inserted in the upper part of the auricle over the piece already there and the lower piece in the lower half of the ear. The ear was encased in a double mould of modeling compound. The incision was sutured lightly as before and wicked. The wound did so well that the wick was withdrawn early.

This case shows, therefore, that cartilage can be transplanted in the ear, even transplanted into a septic cavity, and that it will for the greater part remain. The next point of interest in the case is the condition of the ear today. It is now four months after the insertion of the cartilage. By the way, the acute middle ear suppuration which the tonsillitus set up in the left ear went on to mastoid involvement and required operation, thus filling the patient's cup

of bitterness to the brim. The case lasted over into the service of Dr. Jack and he kindly spelled me by performing the operation. But to return to the condition of the right ear at the present time. Frankly, from the artistic standpoint it is not much to look at, but it is an ear and stands up on the side of the head instead of having crumpled into a distorted mass like a withered leaf. The ear has shrunken



and lost one-third of its vertical diameter and one-eighth of its horizontal diameter, measuring the latter at the top of the canal. The helix has disappeared and the scaphoid fossa has been obliterated and thickened. In short, the ear is much deformed, but as I said before it is still an ear. The young lady has beautifully abundant auburn hair, of which she is very proud and which she arranges most extravagantly. She dresses it in the present fashion down over her ears and adds many touches of her own. Her

other ear is very large and mannish and so looks much better partly covered. No one would be interested, therefore, in either from the standpoint of beauty, but the operated ear is, to me at least, scientifically attractive.

Modelling compound has been spoken of as a splint for the ear. This material I have found so useful that I am tempted to say a word more about it. As you know, it is a spruce gum compound which dentists use for taking impressions of the teeth. The mould which it gives is not so accurate as that obtained with plaster, but it is accurate enough for work about the ear and it is far easier to manipulate. I first used it in cases of orthematoma. In using it for such a purpose a bed is first made for the ear by fitting a crescentic piece of the modelling compound behind the auricle. A larger piece is then softened and with this an impression made of the front of the ear. When this has been hardened by immersing in cold water, the ear is splinted between the two moulds and bandaged to the side of the head. One can hardly imagine a more accurate method of obtaining pressure upon the auricle. A modification of the modelling compound splint is also very useful for holding the auricle when it tends to sag unduly. This occasionally happens after a radical operation in which the incision runs above and in front of the auricle and through the temporal muscles. In such a case, the full splint need not be used, only the front piece. In order to make a splint for this purpose a conical mass of the compound is softened and fitted into the scaphoid and the triangular fossae. This gives it a grip on the ear. The splint does not extend below the top of the canal so that drainage is not interfered with. The lower edge of the splint is built out from the surface of the ear one-half to three-quarters of an inch in order that the bandage may be made to catch under it and pull the auricle upward, backward and inward.

To return to the implantation of cartilage. It should be useful for bettering birth deformities of the auricle. Such cases, of course, must have a considerable amount of soft

tissue with which to work. A little boy of two came to me recently with such a deformity of the left ear and is now waiting his turn for a hospital bed. In one way a case of this kind is more promising than the case which has just been described because the cartilage is placed in a clean cavity. So far in transplanting cartilage under the skin of the bridge of the nose very few of my cases became infected and in the infected wounds the cartilage was only partially lost. My experience has led me to consider that septal cartilage is less liable than rib cartilage to infection and that it resists absorption better. Another advantage which cases of birth deformities possess is that the ear can hardly be made worse. The parents therefore are grateful for any improvement and a word or two of warning should prevent them from expecting the impossible.

The lessons to be drawn from the case here reported, if lessons is not too large a word, are as follows: I waited too long before attacking the abscess radically. As a result more cartilage was destroyed than should have been. My only excuse, and it is a poor one I confess, is that this case is the only one where I have had chondritis of the auricle follow an operative procedure. I caused it, so to speak, and in such happenings there is a strong tendency to wait for nature and not to hurry surgery. The usual signs of extensive chondritis were masked somewhat by the drainage of the pus into the canal. The pus was credited to the radical cavity. Too much cartilage was placed in the upper part of the auricle. What was needed here was a large thin piece of cartilage, not two small pieces placed one over the other like two sheets of writing paper. Having admitted my shortcomings I will quickly come to the main point of the case. Cartilage can be implanted in the auricle and take the place of cartilage destroyed by suppuration. Most of this will remain and it will lessen the deformity which would result if the loss of the original cartilage was not made up. Lastly, the case shows that the feeling of helplessness with which the physician formerly treated these cases should be in the future less.

THE VALUE OF NASO-PHARYNGEAL SURGERY IN THE TREATMENT OF CHRONIC EXUDATIVE OTITIS MEDIA.

By H. O. REIK, M. D., Baltimore, Md.

That chronic exudative otitis media, with its characteristic tendency to progressive deafness, has for its principal cause and continuous exciting factor some abnormality in the nose, pharynx or naso-pharynx—such as hypertrophied turbinates, deflected septum, hypertrophied or submerged diseased tonsils, or adenoids—is an accepted fact, probably settled beyond dispute. The relationship between the naso-pharyngeal condition and the commencing or progressing middle ear disease seems to have been thoroughly established and requires no special argument at this time. It is also an accepted fact that the early correction of such abnormalities is good prophylactic medicine in that it tends to prevent the occurrence of affections of the middle ear. Furthermore, it seems to be the general belief that even after an acute exudative otitis media is established the ear can be restored to normal and safeguarded for the future by prompt and proper treatment of the exciting factors in the nose or throat.

Assuming all this to be true, the possibility of curing or relieving an exudative otitis media that has reached the chronic stage, by removal of the naso-pharyngeal trouble, is not the subject of such universal agreement. As a matter of fact there exists an attitude of skepticism in regard to any and all treatment of chronic exudative otitis media, on the part of the profession as well as of the public. There is abroad a false notion that this diagnosis always carries with it a hopeless prognosis as regards the auditory function and that the unfortunate patient is necessarily condemned to ultimate deafness, the rate of progress downward being more or less rapid according to circumstances. We must admit that there are many persons suffering with

this affection for whom we can do little or nothing either in the way of curing the disease or of preventing its steady advance, but, I am sure there are many more to whom the optimistic, skilled and patient attendant can render most helpful service. It is, however, not within the province of this paper to discuss the various forms of treatment that may be useful under the different conditions observed nor even to offer a classification of these variations. The single topic for present consideration might be embraced in the question: What effect upon the ear can one logically expect from naso-pharyngeal surgery when the chronic exudative otitis media is believed to have had its origin in or to be still excited by abnormal conditions in the nose and throat?

I was led to ask myself this question several years ago for the following reasons: In the first place, I had found that some observers were doubtful of securing beneficial results to the ear from these operations and their doubts were based upon practical experiences rather than disbelief in the theories upon which the treatment rested; for instance, cases which had presented every evidence of relationship between the aural and naso-pharyngeal affection had not been improved nor relieved, in so far as the ears were concerned, by the operation performed. As I shall attempt to show later, I think that many of these failures can be explained and that the doubts, though justified, were misplaced. Secondly, I desired to determine for myself a proper line of conduct toward these patients. Granting the existence of some naso-pharyngeal abnormality in association with a chronic otitis showing advancing deafness, what right had I to advise an operation within the nose or throat, when possibly these organs were not directly suffering, and, what hope could I honestly and conscientiously hold out to the patient that such an operation would benefit his aural condition?

With a view to arriving at a definite answer to these questions, I have during the past few years carefully studied a series of such cases, and it is to some conclusions drawn from

these observations that I particularly wish to ask your attention. The small series of 34 cases upon which I shall report is taken entirely from my private practice; I have not attempted to study the records of those hospital and dispensary cases that were treated during the same period of time because it is so difficult to follow up such cases in order to observe results and to keep the records accurate. In character, the cases treated of here are limited strictly to simple, chronic, exudative (catarrhal) otitis media associated with and believed to be dependent upon some naso-pharyngeal abnormality. No case is included which presented other complications, that is, such as involvement of the auditory nerve, otosclerosis, or the history of hereditary deafness.

A comparison of the conditions before and after treatment must, unfortunately, depend upon subjective tests and it is quite difficult to establish a test which shall be reasonably accurate and reliable and not subject to variations due to the personal equation of either the examined or the examiner. In every case the routine hearing tests were made, but most of these are of little value for *comparison*, with the object of answering the question propounded. The important facts to ascertain were, whether or not the hearing was improved, remained stationary or had become worse immediately after the operation (meaning within two weeks), and, what the relative condition of hearing would be at a considerably later period. In looking over my records it still appears to me that the one and only test that could be utilized is one that I early came to look upon as the most practical and which might be called a self-controlled tuning-fork test. This fork, devised by Dr. Clarence J. Blake and manufactured at the Lawrence Scientific School of Harvard University, is of the middle register (C^2=512 v. s.). It is set in action by drawing the prongs between the thumb and forefinger of one hand so that the tips are brought together by this pressure and spring apart of their own volition as they escape from pressure. The particular fork which I have used, and which has been re-

peatedly tested, can be heard by the normal ear for exactly 20 seconds when held at a distance of one inch from the meatus. It seems to me that this method is not open to the objection that applies to other means of setting a tuning fork in motion, such as striking it upon some object or with some form of hammer. No matter how much pressure is exerted the prongs can only be brought into contact, and no matter how quickly released they can only spring apart: it is therefore scarcely, if at all, subject to any control upon the part of the examiner.

Perhaps it should be mentioned, too, if merely as a matter of acknowledgement that what might be called a theoretical improvement in hearing is not necessarily one of practical value. For instance, ability to hear a moderately high pitched tuning fork for a few seconds longer after treatment may not mean any greater ability to hear conversation. I used the test only as being the most reliable practical test of scientific value and believe that it fully serves the purpose here of *indicating the direction of progress* of the aural disease.

The accompanying table sets forth: the age of the patient: the abnormal condition observed in the nose: pharynx or naso-pharynx: the duration of time in which the deafness had been increasing; the state of hearing by air conduction for the abovenamed fork, expressed in fractions, the denominator representing the time that the fork should be heard by the normal ear, and the numerator showing the time that it was actually heard: the operation performed: the immediate effect upon the hearing, according to the same test made within two weeks of the date of operation: and the condition of hearing, according to the same test, at the latest period thereafter that the patient was seen—which varied from six months to five years. The complaint in every case was increasing deafness and the period of time during which this had been observed varied from six months to fifteen years. Curiously enough this longest existing trouble (Case No. 25) was one of those showing most marked improvement as the result of an operation.

No.	Age	Naso-Pharyngeal Abnormality.	Duration of Increasing Deafness	Hearing for Fork C^2 (512 v. s.)	Operation	Immediate Hearing Result.	Permanent Hearing Result.
1	39 yrs.	Large Adenoid, obstructing the tubal orifices	Several years	R. 10/20 L. 5/20	Adenoidectomy	R. 13/20 L. 19/20	R. 17/20 L. 12/20 2 yrs. later
2	32 "	Hypertrophied R. Inf. Turbinate with fibrous bands in Ros fossa	Two years	R 8/20 L. 8/20	Turbinectomy and removal of bands	R 10/20 L. 10/20	Same 6 mos. after
3	24 "	Fibrous bands across left fossa of Rosenmuller	Six months	R. 20/20 L. 10/20	Removal of bands	R 20/20 L. 15/20	Same 3 yrs. later
4	35 "	Diseased submerged tonsils	One year	R. 12/20 L. 7/20	Tonsillectomy	R 15/20 L. 10/20	Same 6 mos. later
5	14 "	Hypertrophied Tonsils and Adenoid	One year	R. 20/20 L. 8/20	Tonsillectomy and Adenoidectomy	R. 20/20 L. 16/20	R 20/20 L. 20/20 2 yrs. later
6	14 "	Hypertrophied Tonsils and Adenoid	One year	R. 15/20 L. 10/20	Tonsillectomy and Adenoidectomy	No change	
7	17 "	Submerged Tonsils and Scar tissue	Several years	R. 5/20 L. 5/20	Removal of tonsils and scar tissue	R 10/20 L. 8/20	Same 1 yr. later
8	24 "	Deflected Septum	Three years	R. 20/20 L. 1/20	Submucous resection	R 10/20 L. 5/20	Same 1 yr. later.
9	18 "	Hypertrophied Turbinates	Eight months	R. 15/20 L. 15/20	Turbinectomy	R 20/20 L. 20/20	Same 6 mos. later.
10	19 "	Hyper. Right Inferior Turbinate	Two years	R. 10/20 L. 15/20	R Infer. Turbinect	R. 14/20 L. 17/20	R 17/20 L. 20/20 6 mos. later.
11	38 "	Deflected Septum	Six months	R. 15/20 L. 12/20	Submucous resection	R 19/20 L. 19/20	Same 1 yr. later.
12	24 "	Adenoid tissue about tubes	Five years	R. 7/20 L. 7/20	Adenoidectomy	R 10/20 L. 10/20	Same 6 mos. later
13	21 "	Intumescent R. Inf. Turbinate, Hypertrophied L. Middle Turbinate	Three years	R 20/20 L 5/20	Middle Turbinect	R 20/20 L. 9/20	Same 5 yrs. later.
14	33 "	Hypertrophied Middle Turbinates	Several years	R. 12/20 L. 8/20	Turbinectomy	R. 15/20 L. 8/20	Same 2 yrs. later.
15	18 "	Hypertrophied Tonsils	Two years	R. 12/20 L. 10/20	Tonsillectomy	R 14/20 L. 15/20	Same 7 mos. later
16	26 "	Hypertrophied Inf. Turbs. and Adenoid	Eight years	R 0/20 L. 12/20	Infer. Turbinectomy and Adenoidect	No change	
17	32 "	Tonsil masses, previously burned	Three years	R 17/20 L. 5/20	Tonsillectomy	R 17/20 L. 9/20	Same 1 yr later
18	31 "	Hypertrophied Tonsils	One year	R. 15/20 L. 20/20	Tonsillectomy	R 20/20 L. 20/20	Same 1 yr later
19	29 "	Submerged Tonsils	One year	R. 17/20 L. 10/20	Tonsillectomy	R 20/20 L. 13/20	Same 6 mos. later
20	18 "	Hypertrophied Tonsils and Adenoid	Four years	R. 20/20 L 8/20	Tonsillectomy and Adenoidectomy	R 20/20 L. 15/20	Same 1 yr later
21	25 "	Adenoid	Seven years	R. 18/20 L. 12/20	Adenoidectomy	R 18/20 L. 18/20	Same 6 mos. later
22	16 "	Hypertrophied Tonsils and Adenoid	Twelve years	R 5/20 L. 7/20	Tonsillectomy and Adenoidectomy	R 15/20 L. 16/20	Same 2 yrs. later
23	27 "	Diseased submerged Tonsils	Several years	R 8/20 L. 10/20	Tonsillectomy	R. 18/20 L. 15/20	Same 6 mos. later
24	18 "	Hypertrophied Turbinates	Several years	R. 10/20 L. 6/20	Infer. Turbinectomy	R 15/20 L. 10/20	Same 6 mos. later
25	26 "	Hypertrophied Middle Turbinates	Fifteen years	R 6/20 L. 6/20	Middle Turbinectomy	R. 18/20 L. 15/20	Same 6 mos. later
26	32 "	Submerged right tonsil and Hypertrophied R Inf Turbinate	Four years	R 10/20 L 12/20	Tonsillectomy and Turbinectomy	R. 20/20 L. 20/20	Same 3 yrs. later.
27	39 "	Intumescent Inferior Turbinates	Several years	R 15/20 L. 10/20	Cauterization of Infer Turbinates	R 17/20 L. 17/20	Same 6 mos. later
28	33 "	Degenerate Submerged Tonsils	Three years	R. 10/20 L 7/20	Tonsillectomy	R. 19/20 L. 13/20	Same 1 yr later
29	26 "	Hypertrophied Turbinates	Seven years	R 5/20 L 5/20	Cauterization of Turbinates	R 10/20 L 10/20	Same 1 yr. later
30	24 "	Submerged Tonsils	Five years	R. 8/20 L. 15/20	Tonsillectomy	R. 15/20 L. 15/20	Same 6 mos. later
31	19 "	Hypertrophied Tonsils and Adenoid	Two years	R. 15/20 L. 10/20	Tonsillectomy and Adenoidectomy	R 18/20 L. 15/20	Same 6 mos. later.
32	16 "	Hypertrophied Turbinates	Two years	R. 10/20 L. 10/20	Turbinectomy	R 15/20 L. 18/20	Same 6 mos. later
33	28 "	Hypertrophied Tonsils	Thirteen years	R. 15/20 L. 7/20	Tonsillectomy	R 18/20 L. 10/20	R 15/20 L. 10/20 2 yrs. later.
34	29 "	Diseased Submerged Tonsils	Ten years	R. 5/20 L. 10/20	Tonsillectomy	R 8/20 L. 7/20	R 5/20 L 7/20 2 yrs. later.

Examination of this chart will show that in 32 cases there was immediate improvement of hearing to some degree; that in two cases there was no apparent change; that in none was there any immediate loss of hearing. A study of the later observations will show that of these 32 cases, 26 remained improved, 4 showed additional improvement and only 2 lapsed back from the first improvement to the previous state of hearing.

From a consideration of these cases I feel fully justified in answering my question with the following statement: Correction of the naso-pharyngeal abnormalities referred to —hypertrophied turbinates, deflected septa, hypertrophied or submerged diseased tonsils and adenoids—in uncomplicated cases of chronic exudative otitis media will almost certainly check the advance of the aural disease, arrest progress of the deafness and, in some instances, will result in improvement of the hearing. It is impossible to predict in any given case that much improvement of hearing will follow, and I am, in consequence, unwilling to hold out to any of these patients the hope of reclaiming any part of the lost hearing power. I usually tell them now that there is a reasonable certainty that further deterioration can be prevented; that there is a possibility, but not a strong probability, of reclaiming some part of what has been lost; and, I usually find they are quite satisfied with the assurance that the disease can be arrested.

Now I should like to revert to a consideration of that part of my first proposition which relates to the reason why so many failures and disappointments have followed naso-pharyngeal operations in chronic otitis media. I am convinced that the explanation lies in incomplete or improperly performed surgical procedures. Our conclusion that there is a definite connection between the naso-pharyngeal lesion, as a predisposing or active factor, and the slowly progressive chronic middle ear affection has for its foundation the belief that these abnormalities in the upper respiratory tract interfere with the normal physiological function of the tympano-pharyngeal tube. It matters not whether the effect

upon the ear is produced by deflection of the air currents from their natural course through the nares, from mechanical obstruction of the mouth of the tube, from vaso-motor disturbances resulting from chronic inflammation, or from impairment of motion in the musculature of the soft palate and tubes, from the otologist's point of view, the removal of these abnormalities must be accomplished in a manner thorough and complete, without injury to neighboring normal structures and with the object of restoring the parts to a normal anatomical and physiological status. Now, of what avail is a nasal operation that results in an adhesion between the septum and the lateral wall? Might better have left that turbinate or spur alone for the nasal condition has not been improved and, most certainly, no good effect upon the ear need be expected. An operation upon the tonsils which results in ablation of the faucial pillars (containing the palato-glossus and the palato-pharyngeus muscles) has, of course, disposed of the tonsils, but it can scarcely be expected to improve the function of the tubal muscles.

During the time that I have been collecting the material embraced in this paper I have also had the opportunity of seeing some other cases that served to throw light upon these points. A young lady whom I had advised to submit to a tonsillectomy reported to me a year later that, in consequence of some tonsil surgery that she had witnessed while serving as a nurse at one of our general hospitals, she had so feared the operation recommended that she had preferred "to have the tonsils burned out by electricity." She had been subjected to nine cauterizations with the result that there was a mass of tonsillar and scar tissue to which the anterior and posterior faucial pillars were completely adherent. Not very long ago another patient returned to tell me that she had taken my advice and had her tonsils removed. That much was true, but it was not the whole truth; incidentally, the faucial pillars, on both sides, had been taken away, the operator making a clean sweep. It has even been seriously proposed recently, by a surgeon in one of our large hospitals, that hemorrhage after tonsillec-

tomy could be prevented (not controlled, mind you, but prevented) by suturing the anterior and posterior pillars together over the fossa as soon as the tonsil is removed. Can you think of anything less likely to benefit the middle ear? I can scarcely conceive of any circumstances requiring such suturing of these pillars; it certainly is not necessary either to prevent or control hemorrhage.

If such surgical work as this is performed upon patients suffering with chronic exudative otitis media it is not surprising that the aural disease is not arrested nor benefitted. Almost, if not quite, as bad as the effect upon the individual victim of such surgery is the fact that such results seriously discredit the work of competent surgeons; the otologist who has not received the right kind of assistance becomes discouraged and discards the only means of helping these patients, while the unfortunate victim goes out to decry all specialists. It goes without saying that I am not criticizing any of that large body of our competent confreres working in this field, but, every one of us knows, that in every city and large town in this country numerous bungling operations of this sort are being performed daily by men without proper surgical training or knowledge of the specialty of rhino-laryngology. We are too frequently seeing or hearing of evil effects from operations in the nose or throat. For instance, of nasal operations that were frightfully painful; of tonsil or adenoid operations attended by profuse or even dangerous hemorrhage; or of some such injurious results as I have previously referred to. Now, any operation within the nose can be rendered absolutely painless if the cocaine be properly employed; and that does not mean using a large quantity, either. There is no reason whatsoever for the loss of more than a few drops of blood, a dram at the most, in a properly performed tonsillectomy. Adenoidectomy is necessarily accompanied by some loss of blood, but, even in this operation, there need be nothing worthy the name of hemorrhage if a gauze tampon be applied and held in position for a short time immediately after removal of the adenoid. In regard to the tonsillar hemorrhages reported,

I am reminded of a phrase I once heard Prof. Halsted use in the course of a lecture, which I cannot quote verbatim, but which was to the effect that hemorrhage in the course of an operation is the sole means the unconscious patient has to rebuke the incompetent or unskillful surgeon.

In conclusion, I desire to set forth emphatically my belief that simple exudative otitis media which is due to abnormal or diseased conditions in the nose or throat can be arrested in its progress by removal of these exciting conditions; that in such cases the progressive deafness can be stopped and further loss of hearing prevented; that in some few cases the hearing power may be materially improved; and, that success of this kind depends, however, upon the proper performance of naso-pharyngeal operations so that there shall be complete and thorough eradication of the abnormality without injury to neighboring normal structures.

TRANSACTIONS

OF THE

AMERICAN OTOLOGICAL SOCIETY,

FORTY-SEVENTH ANNUAL MEETING.

HOTEL CHELSEA,

ATLANTIC CITY, N. J.

MAY 27 and 28, 1914.

VOLUME XIII. PART II.

PUBLISHED BY THE SOCIETY:
MERCURY PUBLISHING COMPANY, PRINTERS,
NEW BEDFORD, MASS.
1914.

Extra copies may be had by addressing the secretary, Dr. John B. Rae, 247 West 70th Street, New York, N. Y., or Mercury Publishing Company, 112 Union Street, New Bedford, Mass.

CONTENTS.

Members of the American Otological Society 257
Minutes of the Forty-Seventh Annual Meeting 262
How the Patient Can Help Himself in Cases of Chronic Catarrhal Otitis Media. By Stephen H. Lutz, M. D., Brooklyn, N. Y 267
Discussion 270
Modified and Conservative Radical Mastoid Operations for Tolerance of Prostheses in Cases of Diminished Hearing After Middle Ear Suppuration. By W. Sohier Bryant, A. M., M. D., F. A. C. S., New York, N. Y 275
Discussion 277
The Importance of Disturbed Metabolism in the Etiology of Secretory Middle Ear Conditions. By J. E. Sheppard, M. D., F. A. C. S., Brooklyn, N. Y 279
Discussion 282
Two Cases of Sarcoma of the Dura Mater Arising in the Vicinity of the Mastoid Process, with Vague Symptoms Simulating Mastoiditis. Operation in Each Case Following Ultimately by Death. By John F. Barnhill, M. D., Indianapolis, Indiana 285
Discussion 295
Further Remarks on the Use of Nitrate of Silver Applied Within the Mouth of the Eustachian Tube for the Relief of Tinnitus. By William C. Braislin, M. D., Brooklyn, N. Y 299
Discussion 301
Removal of Stapes for the Relief of Severe Auditory Vertigo. By Eugene A. Crockett, M. D., Boston, Mass 302
Discussion 308
The Treatment of Accidental Wounds of the Dura During Operations Upon the Mastoid Process. By Edward Bradford Dench, M. D., New York 310
Discussion 316
The Orbital Approach to the Cavernous Sinus. By Harris Peyton Mosher, M. D., Boston, Mass 318
Discussion 327

Notes on the Use of Radium in Treatment of Diseases of the Ear. By W. Sohier Bryant, A. M., M. D., F. A. C. S., New York, N. Y. 330
Discussion 339
Various Otitic Facial Paralyses. By B. Alexander Randall, M. A., M. D., Philadelphia, Pa. 341
Discussion 346
The Surgical Anatomy of the Temporal Bone. A Lantern Demonstration. By George E. Shambaugh, M. D., Chicago, Ill. 348
Discussion 349
A Further Report of Cases of Inflammatory Affections of the Labyrinth. By Wendell C. Phillips, M. D., New York, N. Y. 353
Discussion 362
Aural Complications in Typhoid Fever. By Edgar M. Holmes, M. D., Boston, Mass. 366
Discussion 374
Congenital Bilateral Microtia with Total Osseous Atresia of the External Auditory Canals, Operation and Report of Cases. By John Randolph Page, M. D., New York, N. Y. 376
Discussion 389
Report of Cases of Aural Infection with the Streptococcus Capsulatus. By Charles E. Perkins, M. D., New York, N. Y. 391
Discussion 396
The Relation of the Labyrinth to the Cerebellum and the Cerebrum. A Lantern Demonstration of Photographs. By John Gordon Wilson, M. D., and F. H. Pike, M. D. 398

MEMBERS

OF THE

AMERICAN OTOLOGICAL SOCIETY.

Names of deceased members while in membership in full face.

HONORARY MEMBERS.

Prof. A. Graham Bell.....1336 19th St.....Washington, D. C.....
Dr. Albert H. Buck.....Hewetts .....Long Island, N. Y....
Dr. E. H. Clarke.
Dr. J. Orne Green.....267 Newbury St.....Boston, Mass.....
Dr. Edw. L. Holmes.
Dr. Hermann Knapp.
Sir William Macewen.....3 Woodside Crescent.....Glasgow, Scotland....
Dr. Arthur Mathewson.....Washington, D. C....
Prof. A. F. Mayer.
Prof. Adam Politzer.....1 Gongazagesse Sq.....Vienna, Austria.....
Dr. Thomas R. Pooley.....40 East 41st St.....New York N. Y.....
Dr. Jonathan S. Prout.....Garden City, L. I....
Dr. H. Luc.....54 Rue de Varenne.....Paris, France.....
Dr. Samuel Theobald.....Cathedral & Howard Sts...Baltimore, Md.....
Dr. E. Williams.

MEMBERS.

Dr. Frank W. Abbott.
Dr. Alpheus E. Adams.....Balmville, Cor. North St...Newburg, N. Y.....
Dr. John L. Adams.....38 East 51st St.....New York, N. Y.....
Dr. C. R. Agnew.
Dr. W. F. Akin.
Dr. Henry A. Alderton.....915 Channing Ave.....Palo Alto, Cal.....
Dr. H. P. Allen.
Dr. Alfred M. Amadon*....543 Boylston St.....Boston, Mass.....
Dr. Emil Amberg*.....270 Woodward Ave.....Detroit, Mich.....
Dr. Joseph A. Andrews.....Santa Barbara, Cal...
Dr. Stephen C. Ayres.....4 West 7th St.....Cincinnati, Ohio.....
Dr. Gorham Bacon.....47 West 54th St.....New York, N. Y.....
Dr. Robert Barclay.....3894 Washington Boulevard.St. Louis, Mo.....
Dr. John F. Barnhill.....Rms. 404-411 Pennway Bldg.Indianapolis, Ind.....
Dr. Edwin W. Bartlett.
Dr. T. Passmore Berens....35 Park Ave.....New York, N. Y.....
Dr. Herbert S. Birkett.....252 Mountain St.....Montreal, Canada.....

Dr. Hugh B. Blackwell*...148 West 58th St.....New York City.....
Dr. Clarence J. Blake.....226 Marlborough St.....Boston, Mass.....
Dr. Frank J. Blodgett.....140 West 72nd St.....New York, N. Y.....
Dr. Charles R. C. Borden*..484 Beacon St.....Boston, Mass.....
Dr. William C. Braislin....556 Washington Ave.....Brooklyn, N. Y.....
Dr. Joseph H. Bryan.....818 17th St. N. W.....Washington, D. C.....
Dr. W. Sohier Bryant.....19 West 54th St.....New York, N. Y.....
Dr. Ernest U. Buckman....99 South Franklin St.....Wilkes Barre, Pa.....
Dr. Charles H. Burnett.
Dr. Ralph Butler.....1824 Chestnut St.....Philadelphia, Pa.....
Dr. R. Bishop Canfield*.....Ann Arbor, Mich.....
Dr. Franklin P. Capron.....118 Angell St.....Providence, R. I.....
Dr. James B. Clemens.....10 East 71st St.....New York, N. Y.....
Dr. Cornelius G. Coakley...53 West 56th St.....New York, N. Y.....
Dr. William Cowen.....35 East 60th St.....New York, N. Y.....
Dr. Charles N. Cox.....257 Jefferson Ave.....Brooklyn, N. Y.....
Dr. Eugene A. Crockett.....298 Marlborough St.....Boston, Mass.....
Dr. J. Frank Crouch.....513 North Charles St.....Baltimore, Md.....
Dr. John P. Davidson.
Dr. Ewing W. Day.....1005 Westinghouse Bldg....Pittsburg, Pa.....
Dr. Edward B. Dench.....15 East 53rd St.....New York, N. Y.....
Dr. J. H. Dix.
Dr. Ernest E. Doble.....52 Cross St.....Quincy, Mass.....
Dr. Arthur B. Duel.....27 East 57th St.....New York, N. Y.....
Dr. John Dunn*.....314 East Franklin St.....Richmond, Va.....
Dr. E. Dwyer.
Dr. Wells P. Eagleton.....15 Lombardy St.....Newark, N. J.....
Dr. E. P. Ely.
Dr. Joseph B. Emerson.....40 East 41st St.....New York, N. Y.....
Dr. George F. Fiske.....100 State St.....Chicago, Ill.....
Dr. Edw. Fridenberg.
Dr. Percy Fridenberg.....60 East 58th St.....New York, N. Y.....
Dr. Harry Friedenwald....1029 Madison Ave.....Baltimore, Md.....
Dr. Biancome E. Fryer.
Dr. Max A. Goldstein*.....3858 Westminster Place...St. Louis, Mo.....
Dr. John Green.
Dr. Emil Gruening.
Dr. George W. Hale.....Wilcox Building.....Nashville, Tenn.....
Dr. Thomas H. Halsted.....831 University Block.....Syracuse, N. Y.....
Dr. Philip Hammond.....483 Beacon St.....Boston, Mass.....
Dr. Herbert Harlan.....516 Cathedral St.....Baltimore, Md.....
Dr. Thomas J. Harris.....104 East 40th St.....New York, N. Y.....
Dr. David Harrower.....9 Elm St.....Worcester, Mass.....
Dr. N. Darrell Harvey.....114 Watermann St.....Providence, R. I.....
Dr. William H. Haskin.....40 East 41st St.....New York, N. Y.....
Dr. R. Johnson Held*.....616 Madison Ave.....New York, N. Y.....

Dr. Neil J. Hepburn.....317 West 23rd St.....New York, N. Y.....
Dr. James Hewitt.
Dr. Christian R. Holmes....8 East 8th St.....Cincinnati, Ohio.....
Dr. Edgar M. Holmes*.....531 Beacon St.....Boston, Mass.....
Dr. Erastus E. Holt.....723 Congress St.....Portland, Me.....
Dr. Frank T. Hopkins*....182 West 58th St.....New York City.....
Dr. Thomas Hubbard*.....515 Nicholas Bldg.....Toledo, O.....
Dr. Frederick L. Jack.....215 Beacon St.....Boston, Mass.....
Dr. Walter B. Johnson.....170 Broadway.....Paterson, N. J.....
Dr. Richard Jordan.....165 West 58th St.....New York, N. Y.....
Dr. Joseph A. Kenefick.....78 East 54th St.....New York, N. Y.....
Dr. B. R. Kennon.....64 Granby St.....Norfolk, Va.....
Dr. Philip D. Kerrison.....58 West 56th St.....New York, N. Y.....
Dr. Gordon King.
Dr. Charles J. Kipp.
Dr. D. Braden Kyle.....1517 Walnut St.....Philadelphia, Pa.....
Dr. J. W. Lawton.
Dr. George A. Leland.....354 Commonwealth Ave....Boston, Mass.....
Dr. John C. Lester.....432 Madison Ave.....New York, N. Y.....
Dr. Eugene R. Lewis*.....1130 Main St.....Dubuque, Iowa .....
Dr. Robert Lewis .....48 West 40th St.....New York, N. Y.....
Dr. W. S. Little.
Dr. Hanau W. Loeb.....537 N. Grand Ave.....St. Louis, Mo.....
Dr. James E. Logan.....1208 Wyandotte St.....Kansas City, Mo.....
Dr. Francis B. Loring.....1420 K St., N. W.....Washington, D. C.....
Dr. David B. Lovell.....Rooms 407-408 State Mutual Building .....Worcester, Mass.....
Dr. C. J. Lundy.
Dr. Stephen H. Lutz*.....284 Hancock St.....Brooklyn, N. Y.....
Dr. G. Hudson-Makuen*....1627 Walnut St.....Philadelphia, Pa.....
Dr. George B. McAuliffe....616 Madison Ave.....New York, N. Y.....
Dr. James F. McCaw.....Bank & Loan Bldg.....Watertown, N. Y.....
Dr. Read J. McKay.....9th & Market Sts.....Wilmington, Del.....
Dr. James F. McKernon....62 West 52nd St.....New York, N. Y.....
Dr. O. A. M. McKimmie*..1330 Mass. Ave. N. W.....Washington, D. C....
Dr. Cyrus S. Merrill.....27 South Hawk St.....Albany, N. Y.....
Dr. Horace J. Miller.
Dr. W. W. Moreland.
Dr. Henry L. Morse.....112 Marlborough St.....Boston, Mass.....
Dr. Harris P. Mosher*.....828 Beacon St.....Boston, Mass.....
Dr. Willis B. Moulton*.....180 State St.....Portland, Me.....
Dr. Robert C. Myles.....46 West 38th St.....New York, N. Y.....
Dr. James E. H. Nichols.
Dr. Henry D. Noyes.
Dr. J. F. Noyes.
Dr. Francis R. Packard....304 South 19th St.....Philadelphia, Pa.....

Dr. John R. Page*.....127 East 62d St.....New York, N. Y.....
Dr. Charles E. Perkins*....125 West 82nd St.....New York, N. Y.....
Dr. Wendell C. Phillips.....40 West 47th St.....New York, N. Y.....
Dr. Norval H. Pierce.....22 E. Washington St.....Chicago, Ill.....
Dr. Edward M. Plummer....5 Adams St.....Charlestown, Mass....
Dr. Oren D. Pomeroy.
Dr. Barton H. Potts.
Dr. George H. Powers, Jr.*.298 Marlborough St.....Boston, Mass.....
Dr. John B. Rae.....247 West 70th St.....New York, N. Y.....
Dr. B. Alexander Randall...1717 Locust St.....Philadelphia, Pa.....
Dr. Wm. Rankin, Jr.
Dr. J. M. Ray.....Atherton Building.....Louisville, Ky.....
Dr. Richard A. Reeve.....48 East Bloor St.....Toronto, Canada.....
Dr. Henry O. Reik.....506 Cathedral St.....Baltimore, Md.....
Dr. W. Scott Renner.....341 Linwood Ave.....Buffalo, N. Y.....
Dr. George L. Richards.....124 Franklin St.....Fall River Mass.....
Dr. Huntington Richards...St. Paul's School.....Concord, N. H.....
Dr. John D. Richards*.....44 West 49th St.....New York, N. Y.....
Dr. Charles W. Richardson..1317 Connecticut Ave.....Washington, D. C.....
Dr. Stephen O. Richey.....732 17th St.....Washington, D. C.....
Dr. Charles E. Rider.
Dr. F. W. Ring.
Dr. Henry W. Ring.....185 Church St.....New Haven, Conn....
Dr. Edmund C. Rivers.....1632 Welton St.....Denver, Col.....
Dr. Arthur W. de Roaldes...624 Gravier St.....New Orleans, La.....
Dr. Walter Roberts.....1732 Spruce St.....Philadelphia, Pa.....
Dr. William K. Rogers....88 East State St.....Columbus, Ohio.....
Dr. D. B. St. John Roosa.
Dr. John D. Rushmore.....129 Montague St.....Brooklyn, N. Y.....
Dr. T. Laurance Saunders*.43 West 50th St.....New York City.....
Dr. Wm. W. Seely.
Dr. Ralph W. Seiss.....255 South 17th St.....Philadelphia, Pa.....
Dr. S. Sexton.
Dr. George E. Shambaugh..People's Gas Bldg.....Chicago, Ill.....
Dr. John B. Shapleigh.....Grand & Washington Aves..St. Louis, Mo.....
Dr. Warren S. Shattuck, Jr.160 Clinton St.....Brooklyn, N. Y.....
Dr. Henry L. Shaw.
Dr. John E. Sheppard.....130 Montague St.....Brooklyn, N. Y.....
Dr. Burt R. Shurly*.....32 Adams Ave. W.....Detroit, Mich.....
Dr. E. Terry Smith.....70 Cove St.....Hartford, Conn.....
Dr. S. MacCuen Smith.....1429 Spruce St.....Philadelphia, Pa.....
Dr. Sargent F. Snow.....707 University Block.....Syracuse, N. Y.....
Dr. Edmund D. Spear.....100 Boylston St.....Boston, Mass.....
Dr. Horatio N. Spencer.....2725 Washington Ave.....St. Louis, Mo.....
Dr. Selden Spencer*.....2725 Washington Ave.....St. Louis, Mo.....
Dr. Frank Sprague.

Dr. Franklin M. Stephens...19 West 54th St.....New York, N. Y.....
Dr. George T. Stevens.....350 West 88th St.....New York, N. Y.....
Dr. Edward Stieren.....Westinghouse Building....Pittsburg, Pa.....
Dr. Samuel B. St. John.
Dr. George Strawbridge....202 South 15th St.....Philadelphia, Pa.....
Dr. Joseph A. Stucky*.....Lexington, Ky.....
Dr. Edward Blair Sutphen*.162 South St.....Morristown, N. J.....
Dr. Theron Y. Sutphen....999 Broad St.....Newark, N. J.....
Dr. Lewis H. Taylor.....82 South Franklin St.....Wilkes Barre, Pa.....
Dr. J. J. Thomson*.....40 West 47th St.....New York City.....
Dr. William Thomson.
Dr. Robert Tilley.
Dr. Andrew Timberman*...112 East Broad St.....Columbus, Ohio.....
Dr. George L. Tobey*.....416 Marlborough St.....Boston, Mass.....
Dr. J. J. B. Vermyne.
Dr. Ernest de Wolfe Wales..423 Board of Trade Bldg.. Indianapolis, Ind.....
Dr. D. Harold Walker.....390 Commonwealth Ave....Boston, Mass.....
Dr. David Webster.....24 East 48th St.....New York, N. Y.....
Dr. Joseph A. White.....200 East Franklin St.....Richmond, Va.....
Dr. Edward Wheelock.....26 Gibbs St.....Rochester, N. Y.....
Dr. Fred Whiting.....19 West 47th St.....New York, N. Y.....
Dr. Frederick M. Wilson...834 Myrtle Ave.....Bridgeport, Conn.....
Dr. John Gordon Wilson*..104 S. Michigan Ave.....Chicago, Ill.....
Dr. Hiram Woods.....842 Park Ave.....Baltimore, Md.....

*Associate.

American Otological Society.

The forty-seventh annual meeting of the American Otological Society was held at the Hotel Chelsea, Atlantic City, N. J., Wednesday and Thursday, May 27th and 28th, 1914.

The following members and guests registered during the meeting:

Members.

J. H. BRYAN
W. C. BRAISLIN
R. C. MYLES
J. F. BARNHILL
E. M. PLUMMER
F. R. PACKARD
S. MacCUEN SMITH
D. H. WALKER
RALPH BUTLER
T. P. BERENS
B. ALEX. RANDALL
W. C. PHILLIPS
JAS. F. McKERNON
H. O. REIK
J. E. SHEPPARD
F. M. STEPHENS
H. S. BIRKETT
G. E. SHAMBAUGH
J. B. SHAPLEIGH
PHILIP HAMMOND
N. D. HARVEY
T. H. HALSTED
W. SCOTT RENNER
JOHN L. ADAMS
JOHN B. RAE
EDWARD B. DENCH
F. J. BLODGETT
W. S. BRYANT
CHAS. W. RICHARDSON
E. W. DAY
ROBERT LEWIS
E. A. CROCKETT
C. G. COAKLEY
J. M. INGERSOLL
F. L. JACK

Associate Members.

THOS. HUBBARD
H. W. LOEB
H. P. MOSHER
G. HUDSON-MAKUEN
J. J. THOMSON
B. R. SHURLY
O. A. M. McKIMMIE
J. R. PAGE
J. GORDON WILSON
E. M. HOLMES
S. H. LUTZ
SELDEN SPENCER
H. FRIEDENWALD
C. E. PERKINS

Guests.

HENRY L. SWAIN
GEORGE F. LIBBY

Wednesday Morning, May 27th.

Executive Session.

The meeting was opened by Dr. James F. McKernon, President, when the minutes of the forty-sixth annual meeting were read and approved.

The President appointed as the Business Committee, Dr. Jos. H. Bryan, Dr. J. E. Sheppard, and Dr. Herbert S. Birkett.

The Secretary read several communications inviting the Society to hold the 1915 meeting at San Francisco. On motion, this matter was referred to the Business Committee.

The Secretary read a letter from the American Laryngological, Rhinological and Otological Society inviting this Society to appoint a committee of three to consider, in conjunction with similar committees, the question of the Under-Graduate and Post-Graduate instruction of the specialist. On motion this was also referred to the Business Committee.

The Secretary presented the resignations of Dr. Samuel Theobald of Baltimore and Dr. Adolph Alt, St. Louis. On motion, the resignations were accepted.

The Secretary reported the death of Dr. Edwin W. Bartlett of Milwaukee and the death of Dr. John Green of St. Louis, Mo.

The report of the Secretary-Treasurer was submitted and on motion approved.

The following nominations for membership were made:

Dr. J. Ingersoll of Cleveland, O. Proposed by Dr. Chas. W. Richardson; seconded by Dr. Thos. H. Halsted.

Dr. A. F. Koetter of St. Louis, Mo. Proposed by Dr. J. B. Shapleigh; seconded by Dr. Selden Spencer.

Dr. John R. Fletcher of Chicago, Ill. Proposed by Dr. J. G. Wilson; seconded by Dr. W. Sohier Bryant.

Dr. Samuel Theobald of Baltimore, whose resignation had just been accepted, was nominated for Honorary Membership. This nomination was referred directly to the Committee on Membership.

Dr. Dench spoke briefly with regard to the necessity of the Proposers and Seconders of Candidates, writing letters to the Committee on Membership, setting forth *in detail* the claims of such candidates for membership in the Society. He also deemed it advisable that the Proposer and Seconder, as far as practicable, should come from the same section as the candidate, in order that intimate information might be supplied for the guidance of the members of the Committee on Membership.

The Society then adjourned to Scientific Session, when papers were read by Dr. S. H. Lutz, Dr. W. S. Bryant, Dr. J. E. Sheppard, Dr. J. F. Barnhill, Dr. W. C. Braislin, Dr. E. A. Crockett and Dr. E. B. Dench.

Wednesday Afternoon, May 27th.

Scientific Session.

Papers were read by Dr. H. P. Mosher, Dr. W. S. Bryant, and Dr. B. Alex. Randall.

Executive Session.

The President appointed Dr. Jos. H. Bryan of Washington as the Representative to the Executive Committee of the American Congress of Physicians and Surgeons, and Dr. Henry O. Reik of Baltimore as Alternate.

The Business Committee reported that it was their opinion that the question with reference to the meeting in San Francisco be laid on the table. On motion, this was approved.

The Business Committee further recommended that the President appoint a committee of three to confer with the Committee of the American Laryngological, Rhinological and Otological Society with reference to the education of the medical specialist. The President later appointed on this committee, Dr. B. Alex. Randall, Dr. H. S. Birkett and Dr. T. J. Harris.

The Business Committee presented the following nominations for 1914-1915.

For President—Dr. Chas. W. Richardson, Washington.

For Vice-President—Dr. C. R. Holmes, Cincinnati.

For Secretary-Treasurer—Dr. John B. Rae, New York City.

Committee on Membership—Dr. Robert Lewis to serve for three years.

Committee on Publication—Dr. B. Alex. Randall, Dr. Arthur B. Duel, the Secretary, *ex officio*.

All of those nominated were duly elected.

The following excuses for absence were presented by the Secretary: Dr. Geo. L. Richards, Dr. A. M. Amadon, Dr. W. P. Eagleton, Dr. Jos. A. White, Dr. C. N. Cox, Dr. A. B. Duel, Dr. Jos. A. Stucky, Dr. J. A. Kenefick. Dr. Wm. Cowen.

On motion, the excuses were accepted.

The Special Committee on the Cancer Question reported progress.

The Committee on Membership recommended that Honorary Membership be conferred upon Dr. Samuel Theobald of Baltimore. On motion, this was unanimously carried.

The Committee on Membership recommended the election of Dr. Eugene R. Lewis, Dubuque, Iowa; Dr. Geo. H. Powers, Jr., Boston, Mass.; Dr. Geo. L. Tobey, Boston, Mass.; Dr. R. Johnson Held, New York City; Dr. Edward Blair Sutphen, Morristown, N. J.

On motion, these gentlemen were duly elected to membership.

On the recommendation of the Committee on Membership, the following candidates were held over for one year: Dr. Benj. D. Parish, Philadelphia, Pa.; Dr. Wm. E. Sauer, St. Louis, Mo.

Thursday Morning, May 28th.

Scientific Session.

Papers were read by the following: Dr. Geo. E. Shambaugh, Dr. J. Gordon Wilson, Dr. W. C. Phillips, Dr. E. M. Holmes, Dr. J. R. Page, and Dr. C. E. Perkins.

Executive Session.

On motion of Dr. Dench, seconded by Dr. Randall, it was resolved that the assessment for the coming year be ten dollars ($10.00).

The Society then adjourned.

HOW THE PATIENT CAN HELP HIMSELF IN CASES OF CHRONIC CATARRHAL OTITIS MEDIA.

By STEPHEN H. LUTZ, M. D., Brooklyn, N. Y.

This paper is a plea for the better instruction of patients suffering from chronic catarrhal otitis media. They should be instructed and advised in reference to hygiene and diet, methods of ventilation and manner of dress. A careful history of their general condition as well as their ear condition should be obtained. Patients must be impressed with the necessity of giving to the aurist detailed information in regard to their general health. It requires more than a number of visits to the office of the aurist and the after a time more or less perfunctory inflations for patients to obtain the help they are seeking.

Otologists are agreed that diseased conditions of the naso-pharynx and nose and the consequent involvement of the Eustachian tubes are the cause of ear troubles in a large percentage of cases, but in many cases we must go far away from the upper air passages in seeking for our cause. The other regions of the body must not be forgotten. The proper circulation of healthy blood, the function of the liver and kidneys and a healthy large intestine mean much to the patient in our prognosis. Aneurism, tuberculosis, syphilis, the onset of acute disease, anaemia or drug habits must all be remembered. Careful observation and examination and a clear reasoning mind has helped many an aurist over the hurdles that threw his predecessors into the ditches of despair. The otologist who does all his work with a Politzer-bag, catheter or other instruments frequently finds his patients missing when he needs them most.

Woakes was not so far off in his original writings on the association of ethmoiditis with middle ear disease. At the time it was considered preposterous but in following years the influence of diseases of the nose and naso-pharynx on the

middle ear was more plainly shown until Wilhelm Meyer clinched the argument when he published his paper on "Adenoid Vegetations in the Naso-Pharynx."

Abnormal conditions in the nose or sinuses, with the consequent poor drainage, extension of inflammatory processes from these to the tube mouth, obstruction by the presence of adenoid masses or enlarged posterior ends, or by cicatricial remains due to attempts at removal of adenoids, mucus deposited in the Fossa of Rosenmüller, paralyses of, or other interference with the action of, the levator palati and the tensor palati are all causes of the deafness which we call chronic catarrhal otitis media.

A deviated septum may not always interfere with inspiration or expiration, nor will a diseased accessory sinus, of itself, directly, always cause trouble, but either of these causes will accumulate or retain mucus and the effort to dislodge the accumulations will almost certainly involve the ear sooner or later. Any blocking in the nose or naso-pharynx interferes with the normal action of the tubo-palatal muscles and therefore the ventilation and drainage of the tube is impaired.

Pressure at localized points is a cause of circulatory disturbance, particularly in the venous return from the middle turbinate and Eustachian tube, which empty into the veins of the pharynx, and by this imbalance congestion of the tube lining and of the mucous membrane of the drum cavity results, with swelling of the lining and diminished calibre of the tube. Later on, as a result of impaired ventilation and drainage, exhudates form and are retained in the tube and in the cavity of the tympanum and also in the area surrounding the tube mouth. This exhudation congestion in the naso-pharynx causes the formation of masses of mucus which are hawked backward or blown forward. These acts cause rarifaction or pressure in the naso-pharyngeal cavity; exhausted or rarified air in the naso-pharynx allows greater pressure on the drum from without and imbalance is the result and this difference in air pressure is exerted on the tubes and on their control muscles. The trouble begins at this

time from the frequent efforts to clear the posterior nares. Violent blowing of the nose, the suction and pulling to aspirate the mucus backward cause the muscles to stretch, recover, stretch again and this is done repeatedly. Gradually the tube mouth is distorted and becomes a receptacle and retention chamber for infectious laden mucus to further the process already begun.

We have all seen cases in which the effort to dislodge a mass of mucus in order to clear the breathing passage was the beginning of the ear trouble for which the patient sought help.

We know that careful well done nasal surgery will often stop the progress of the ear involvement by providing a fairly natural open air channel with a fair return of muscular control of ventilation of the tympanic cavity.

In various text books we find advice as to methods of clearing the nose. Sprays, douches, blowing the nose with one nostril closed or drawing the mucus back and ejecting it are advised. All of these methods have each and every one had many victims in the past.

Let me beg of you to carefully instruct your patient how to blow the nose. This seems like a simple thing to dwell upon but it will be found by careful practice to be the best means of helping the patient to help himself and retain whatever improvement he may have gained by treatment.

We can trace almost all of our suppurative cases to one of these methods; by pressure against the tube mouth infected material enters the tube and sometimes the tympanic cavity as well.

Instruct the patient to blow gently both nostrils at once into a handkerchief held about an inch away from the face. The fingers must not compress the nose at any place over the yielding soft parts of the nose, in fact they must not touch the nose at all.

I instruct my patients to place the thumb on one side and the forefinger on the other side, at least an inch away from the side walls of the nose, thus supporting the handkerchief like a bag instead of crumpling it in a wad and pressing

firmly up against the nose as the average person does. When a clear pasage of air can be had, stop blowing. There may be some mucus left in the nose but not enough to block the nose and interfere with ventilation.

Some persons do better holding the handkerchief in two hands, one on either side, resting against the cheeks.

Whatever way is used do not allow the least pressure on the nose to interfere with both nostrils being cleared at one and the same time. If the mouth can be slightly opened while blowing there can not be any amount of back pressure toward the Eustachian tubes.

Discussion.

Dr. William C. Braislin, of Brooklyn, N. Y., held that many cases of adenoids are not relieved by the adenoid operation because of the fact that the nose is lacking in development. In such cases the adenoid operation is not sufficient. Proper instruction in the matter of blowing the nose is an important aid in the proper development of the nose as well as in the prevention of adenoids.

Dr. E. B. Dench, of New York, had found, in a number of cases in which, as Dr. Braislin had suggested, the adenoid operation was not sufficient, that a supratonsillar lobe had been left, or had recurred. Removal of this would relieve the symptoms of obstruction.

Dr. Robert C. Myles, of New York, agreed with Dr. Lutz that many cases of infection of the middle ear may be traced to the improper blowing of the nose. In his experience, many cases in which an imbedded tonsil grows upward, under the palate, progressive hardening of hearing had been markedly improved by operation.

Dr. John F. Barnhill, Indianapolis, Ind., thought satisfactory blowing of the nose could not be accomplished in the manner described by Dr. Lutz. He also considered some

of the physiological propositions laid down were incorrect. In his experience there was a thickening and hardening of the muscles and not a laxity. He did not accept the view that adenoid tissue recurs. In cases in which this tissue was said to recur he considered that an imperfect operation had been done. The cause of bad breathing followed adenoid operation was usually due to the fact that there is deformity of the bony structures which make up the parts involved. If there were a good nose the child would breathe properly. With a high arch, deformed cervical spine, or deformed nose it will breathe poorly. If necessary the arch should be widened by an ortho-dental surgeon.

Dr. Burt R. Shurly, Detroit, Mich., thought the paper progressive and interesting, and in line with what might be called the specialty of preventive otology. The matter of blowing the nose, for example, opened up a large field of preventive otology in the direction of the education of patients. The care of the nasopharynx, the proper method of blowing the nose, the methods of increasing the resistance of the individual, the nutritional problems of infancy and childhood, were all along the line of preventive otology, and were some of the matters to be borne in mind in the endeavor to develop stronger constitutions and healthier individuals. An enormous number of correlated problems were involved, among which might be mentioned that of the ductless glands.

Dr. Hanau W. Loeb, St. Louis, Mo., endorsed what Dr. Barnhill had said with reference to adenoid growths and obstruction of the nose. He had had cases in which the obstruction did not depend upon the adenoids but upon deflection of the septum. To the obstruction due to hypertrophy of the turbinate might be added that which is due to the small size of the nose. His own experience had been that despite the removal of every vestige of adenoid tissue there might be a recurrence.

Dr. John E. Sheppard, Brooklyn, N. Y., considered the matter brought up by Dr. Lutz a very important one. Re-

ferring to Dr. Barnhill's objection to the proposed method of blowing the nose, it might be answered that new habits are hard to form, but once the proper habit had been formed in this regard the patient would experience great relief. Should this fail to give relief, further treatment was indicated.

Dr. George E. Shambaugh, Chicago, believes that in connection with the subject discussed in Dr. Lutz's paper, we should keep in mind the importance of instructing patients how to overcome the habit of taking colds. It is true that in America there is an increasing tendency for people to treat themselves more and more like hot-house plants. This tendency is greatly fostered by the use of steam heat, especially in the apartment houses of our cities. Unless care is taken to guard against it people living in steam heated apartments usually find themselves living in rooms much over-heated and too dry. The tendency to suffer from more or less continuous head colds during the winter is not alone due to adenoid growth. People should be taught the importance of keeping the living room cool, rather than too warm, also the necessity of providing ample means of moistening the air of the living room. The importance of sleeping in cold sleeping rooms should also be explained. The ideal sleeping room in any climate for winter as well as for summer is the sleeping porch, which fortunately is coming more and more into general use, especially in our cities. In a word, the whole problem is one of hardening oneself by keeping the living room cool, the bedroom open, and taking cold baths.

Dr. Robert Lewis, New York, challenged Dr. Barnhill's statement with reference to the recurrence of adenoids. The comparison between the removal of adenoids and the removal of tonsils was not a fair one. The faucial tonsil is contained, to a large extent, within a capsule, while the pharyngeal tonsil has no such capsule. After the most thorough and careful removal he has seen adenoids recur in his own prac-

tice, and he has seen them recur following operations performed by thorough and careful operators.

Dr. Joseph H. Bryan, of Washington, D. C., had had adenoid recurrences, or what he considered to be such, and believed that they do recur after very thorough operation. The question might be asked, "What is 'complete removal' of adenoids?" Is it possible to remove all adenoid tissue in the nasopharynx? It was not possible without injury. In order to remove all it would be necessary to go down to the bone, and in that event enough cicatricial tissue would result to do more harm than the adenoids would cause. Sometimes the removal of obstruction was offset by chronic congestion due to continued growth in the adenoid ring. In some cases, when the patient was not doing well, it was not desirable to entail further risk by accomplishing the complete removal of adenoid tissue.

Dr. Sheppard added that he had had adenoids recur after complete removal. Through improper diet and hygiene there was engorgement of all the tissues of the nose, and the same condition occurred in the mucosa of the nasopharynx. He had seen cases in which recurrence, or new growth, of lymphoid tissue had begun, but by putting the patient on a proper diet and instituting proper hygienic conditions it had completely disappeared.

Dr. Barnhill added that he would like to admit that adenoids do not ever return after operation, but his experience was to the contrary—he had had patients with adenoids after he had removed them. He had had occasion, after children had grown up, or several years after he had removed the adenoids, to use the palate retractor, and had found lobes of adenoid tissue. Dr. Loeb had said he removed all the adenoid tissue that could be felt with the finger. His own fingers were not sensitive enough to warrant his feeling sure that he had felt and removed all the adenoid tissue. He did not believe that it was always possible to be

sure of removing every vestige of it. With the tonsil in full view one did not always get it all out. If this were true of the tonsil, which was in full view, it was quite evident that one did not always get all of the adenoid tissue.

Dr. Dench added that he had reference to the operation formerly employed, before the days of enucleation.

Dr. Loeb added that he did not know why lymphoid tissue grows in the pharynx. Throughout the pharynx this tissue is found, and some of that which was left might take on hypertrophy just as had that which was removed. So far as he could detect with the finger it was all removed in cases in which recurrence had taken place.

Dr. Myles, continuing the discussion, said in children under two or three years of age many adenoid follicles were not yet hypertrophied. These would later develop on the sides of Rosenmuller's fossa whether operation had been performed or not. This was a matter of nature, and could not be helped. After removal of part others would grow. The same thing applied to the development of the lingual tonsil after removal of the pharyngeal tonsil.

Dr. Lutz, in closing the discussion, said eruptive diseases, with their inflammatory condition in the nasopharynx, give rise to hypertrophy of existing lymphoid masses, and this might be mistaken for a recurrence of that which had been removed. Gentle curettage of the nasopharyngeal cavity with several thicknesses of gauze would help to clear up a lot of these little masses.

MODIFIED AND CONSERVATIVE RADICAL MASTOID OPERATIONS FOR TOLERANCE OF PROSTHESES IN CASES OF DIMINISHED HEARING AFTER MIDDLE EAR SUPPURATION.

By W. SOHIER BRYANT, A. M., M. D., F. A. C. S., New York, N. Y.

Among cases of residual deafness, following middle ear suppuration, including both those cases that have undergone any form of operation and those that have undergone none, there is a large number of cases in which the hearing is very considerably increased by the use of suitable prostheses. Among these last, there are some cases in which the ears, although they may be quiet and dry, and have no suppuration, begin to suppurate as soon as a prosthesis is applied. This class of ear is characterized by the presence of crevices harboring desquamated epithelium, cicatricial folds, trabeculae and ossicular remnants about the middle ear and mastoid antrum.

In these cases we have found that a modified radical mastoid operation or a so-called conservative radical mastoid operation is indicated. The intention in these operations is twofold: first, the hearing mechanism is to be preserved in as good condition as it is previous to the operation; and second, the middle ear and accessory cavities are to be cleaned so that the middle ear will heal in a smooth, readily epidermatized, clean cavity and will consequently tolerate a prosthesis.

The technic used in these operations is adapted of course to the condition present. In the conservative radical mastoid operation the auricle is deflected forward, together with the membranous canal, in the way preferred for the radical mastoid operation. The posterior and superior walls of the osseous meatus are removed, exposing the attic, the antrum and middle ear precisely as in the radical mastoid operation.

The middle ear is carefully freed from obstructions, such as remnants of the major ossicles, cicatricial bands or walls of trabeculae. Care is taken not to remove the dermoid covering of any structure that is not also to be removed; that is, unnecessary denudation of the middle ear should be avoided. After the middle ear has been carefully freed of the unnecessary contents, the membranous canal and concha are treated plastically, to insure an enlargement of the external meatus, in proportion to the enlarged osseous meatus. The modified radical operation follows the same technic as the preceding except that the major ossicles may be retained in position.

After these operations the ear usually heals rapidly and gives very satisfactory results in hearing by the tolerance of a prosthesis. The following cases illustrate the practical value of these operations. The first patient, a woman 33 years of age, was dependent for all her hearing on a chronic suppurating ear. Without a prosthesis the Politzer acoumeter was heard at 7 inches; with the prosthesis in position, the acoumeter was heard at 25 inches.

The suppuration ceased soon after the cleansing treatment was commenced. The Politzer acoumeter was then heard at 6 inches without the prosthesis and at 23 inches with the prosthesis. Soon after a continuous use of the prosthesis, the suppuration recurred but was easily controlled. This procedure was repeated several times and always with the same results. It was then decided that a modified radical mastoid operation would be performed on the ear in order to preserve what hearing remained, and, at the same time, make the ear tolerant of a prosthesis.

The condition of the middle ear was as follows: the membrana tympani had largely disappeared and the remnants of the ossicles were bound in a cicatrix in the posterior region of the tympanic cavity. Since the hearing was very satisfactory with a prosthesis in position, it was decided not to risk any impairment of the hearing by interfering with the contents of the tympanic cavity. The antrum was opened widely into the tympanic space and canal and this was suffi-

cient to assure cleanliness enough to allow the continuous use of the prosthesis.

The operation followed the technic of the ordinary radical mastoid operation, as far as the management of the membranous and osseous meati was concerned. Nothing in the tympanic cavity was removed and great care was exercised not to disturb the remnants of the malleus and stapes, which was all that remained of the ossicular chain.

The ear healed by first intention and in a few days the ear and canal were dry and desquamating. Two years after the operation, the Politzer acoumeter was heard at 15 inches without the prosthesis, and at 96 inches with the prosthesis in position.

The other case was similar to the previous one. It was thought best, however, to remove the remnant of the major ossicles, and therefore a conservative radical mastoid operation was performed. In this case, the operation included the removal of the remnants of the malleus and incus and part of the head and crura of the stapes. The results were quickly obtained and were as satisfactory as in the first case.

LITERATURE.

The Use of Aids to Hearing in Middle Ear Deafness. Jour. of Laryngology, Rhinology and Otology. Vol. 29, Jan. 1914, p. 12.

The Protective Mastoid Operation the Operation of Election. Surgery, Gynecology and Obstetrics, Dec. 1913, p. 753-755.

Discussion.

Dr. E. B. Dench, New York, emphasized the important point of not curetting the mucous membrane of the middle ear in every case. At one time he thought it necessary to thoroughly curet the entire tympanic cavity, but in later operations he had not curetted the antrum. He cited a case in which a man had undergone a radical operation upon one ear in Vienna, subsequently coming to him with cholesteatoma in the tympanic vault of the opposite side of an old otitis media. He would have cut down the hearing con-

siderably if he had done the complete radical operation. He confined the radical operation to the upper part of the cavity, cleaning out the cholesteatoma, and the man went away with very good hearing. He thought there was a decided place for this partial radical operation to which Dr. Bryant had directed attention.

Dr. Bryant, in closing the discussion, said a prosthesis is a mechanism which is used to replace a lost part. He did not use "artificial membrane" because these structures were not in any sense of the word artificial membranes.

THE IMPORTANCE OF DISTURBED METABOLISM IN THE ETIOLOGY OF SECRETORY MIDDLE EAR CONDITIONS.

By J. E. SHEPPARD, M. D., F. A. C. S., Brooklyn, N. Y.

At the 1905 meeting of the American Laryngological, Rhinological, and Otological Society, I read a paper entitled "Otitis Media Mucosa," in which I attributed the condition to a variety of causes, amongst others "cold in the head, nasal polypi, hypertrophied third and fifth tonsils, blowing the nose too hard, nasal douche, washing the hair, etc.," and to vasomotor rhinitis in only one case. In the rather general discussion which followed, the late Dr. Sprague attributed "many of these cases to some vasomotor disturbance, or disease of the nervous system," adding "there is usually some constitutional condition, such as disease of the stomach, liver or kidneys." Dr. Richardson said "a good many of these cases are due to vasomotor disturbances, and constitutional treatment is indicated." From Dr. Stucky's remarks I quote "the disease is not primarily a pathologic condition, but symptomatic." The general health, especially the gastro-intestinal canal, should be thoroughly investigated and regulated." I wish to give credit to these three gentlemen, and especially to Dr. Stucky, for having thought further along the etiologic line in this condition than I at least, and perhaps than most of us, had done at that time.

In the present paper I wish to extend what I have to say to a somewhat larger field, to include many of the acute tubal conditions with or without the outpouring of more or less serous fluid into the tympanic cavity; the more purely Otitis Media Serosa, without marked involvement, at least when we see them, of the Eustachian tubes; the cases of Otitis Media Mucosa; and most of those cases, perhaps all, in which the predominating symptom is a more or less profuse mucous, or muco-purulent, discharge from the ear. It is my present belief that all these cases of secretory middle ear

conditions may be attributed to some vasomotor disturbance, usually a paralysis or paresis, involving in varying degrees the mucosa and underlying vascular areas of the nose, nasopharynx, Eustachian tube and tympanic cavity. In passing, I should like to emphasize the importance from the viewpoint of therapeutics of distinguishing clearly between this condition and a genuine hypertrophy of these structures. This vasomotor paralysis, or paresis, I believe to be in its turn an evidence, or expression, of some general toxemia acting upon the sympathetic nervous apparatus, and due, much more often than anything else, to a condition of disturbed metabolism, and insufficient elimination.

Gould's definition of metabolism is "the power that organized bodies possess of continuously using up and renewing the matter composing the body." By the term "disturbed metabolism" I wish to include in a rather broad general sense those abnormal conditions due to the amount we eat,—to what we eat, and how we eat it, as well as to what we drink, and when we drink it; and by "insufficient elimination" I mean to include that by the bowel, the kidneys, and the skin.

Metabolism is easily disturbed by over eating, even of perfectly proper kinds of food. It is being more and more recognized, though not so generally acted upon, that mankind eats too much. By way of example, one of the most frequently observed errors is the addition to three abundant meals of a pint, a quart, or even more of milk—often sufficient milk of itself for a person to live on without the addition of one, to say nothing of three, meals a day.

A perfected metabolism depends undoubtedly to a great extent upon *what we eat.* Disturbance thereof arises much more often, in our experience, from excess of the *protein* foods, whether animal or vegetable, though oftener the former, than of the *carbo-hydrates,* although this latter group of foodstuffs would appear to be responsible for some of the disturbance, and more often in children than in adults. To cut down the supply of proteins, it becomes necessary to limit the supply of meats, particularly the red meats,

meat-stock soups, eggs, milk, cream, and butter, among the usual articles of food, and for excess of the carbo-hydrates to limit the sugars and starches, by forbidding candy and excess of sugar in any form, by limiting potato, and the substitution of whole-wheat bread for the almost universally-used white bread.

Under the heading of *how we eat it*, I wish particularly to emphasize the importance in this class of cases of proper, meaning thereby thorough, mastication of food. To accomplish this most practically, I insist upon my patients eating their meals dry, all fluids being forbidden from ten minutes before until a half-hour after eating.

With regard to *what we drink*, and *when we drink it*,—the latter part of the proposition has just been answered, and can be summed up in the following phrase, "as little as possible at meals, and as much as possible between meals." As to what our patients in this condition may be permitted to drink, it certainly is true that the nearer they come to limiting themselves to water the better, and it is of great importance to curtail to a minimum the use of alcoholics, and especially so their daily use, even in small quantities. Milk should not on any account form a regular part of the diet of those who eat their ordinary three meals, and no one will, I think, claim that tea and coffee, as ordinarily used, are conducive to a perfect metabolism.

Insufficient elimination, whether by bowel, kidney, or skin, is a most important factor in the upsetting of the metabolic process.

I feel that I perhaps owe you an apology for having occupied so much of your time with what is at best, but a very imperfect and incomplete outline of what is required to accomplish the desired results in the class of cases under discussion. At the same time, I have been forced by many unhappy experiences to a realization of the fact that, unless we ourselves take up this end of the treatment, and act upon a proper interpretation of the picture presented by the parts with which we have to deal, we will fail to obtain results.

I might, but will not, relate to you the history of many cases that have demonstrated conclusively the futility of purely local treatment, or for that matter of any local treatment, for the achievement of permanently satisfying results in the so-called secretory middle ear catarrhs, thus completely reversing myself in what I had to say in the paper referred to above, in which I advocated repeated incision of the tympanic membrane as the only treatment that had at that time given results that were even partially satisfying.

I should like to recall to your minds the various pronouncements on this subject by our colleague, Dr. Stucky, in so far as it concerns our specialty, and, as showing the far-reaching effects of disturbed metabolism on other organs and parts of the body, and as very suggestive to us, I would like to direct your attention to articles read by Dr. Plönies, of Hanover, Germany, and by Dr. L. F. Bishop of New York at the International Congress of Hygiene and Demography, held in September, 1912, at Washington, D. C., and published in Volume II. Part I., of their Transactions.

Discussion.

Dr. Stephen H. Lutz, Brooklyn, N. Y., was glad the reader of the paper had gone into details regarding the kinds of food to eat, etc. Excellent results could be obtained from proper instructions to patients. He was in the habit of telling them to eliminate butter, milk, and meat from the diet. He had been convinced for a long time that spring and fall colds are largely due to disturbed metabolism. In such cases rhubarb and soda, and ox-gall and pancreatin were good. It was difficult for the otologist to take up the question of medication, but it was practically useless to send these patients back to the family physician, and less useful to send them to a gastro-enterologist. He mentioned a physician in Brooklyn who treats the majority of his patients by dieting them. They gradually drift away from him in time, but would all admit that they were improved while they persisted with the diet.

Dr. S. MacCuen Smith, Philadelphia, confirmed Dr. Sheppard's findings. He had had cases of non-infectious accumulations of fluid in the middle ear (hydrops ex vacuo) which were unquestionably due to faulty metabolism. He had found a number of these cases in women who smoke. The particular symptoms he had been called upon to relieve were tinnitus aurium and a feeling of fulness in the ear and in many instances relief was obtained by careful dieting, without other treatment.

Dr. Burt R. Shurly, Detroit, Mich., thought Dr. Sheppard had obtained the results reported by stepping over the line between internal medicine and otology. A thorough understanding of the psychology of the individual was the basis upon which these problems must be solved, the intricacies of the gastrointestinal tract, of course, being well understood. All of the cases reported in the paper concerned patients around forty years of age, a period when the destructive processes are more active than the constructive. Calomel and soda are well recognized as useful, and should not be forgotten in these cases. Colonic irrigation, too, was apt to be forgotten, despite the fact that its usefulness is well known. There was always danger of overstepping the mark and becoming dietetic cranks.

Dr. Robert C. Myles, New York, corroborated what Dr. Sheppard had said with reference to the remarkable results obtained by careful diet and hygiene in certain cases of tinnitus aurium. He had been observing this for six years, especially since Dr. Stucky called attention to the matter. In 1908 he had begun to use liquid vaseline and its products very freely. He referred many of his patients to the gastroenterologist, with favorable results in some cases.

Dr. E. B. Dench, New York, called attention to the causative role played by the excessive use of sodium chloride, as pointed out by Lermoyes, in an article published some years ago. Alcohol, in moderate amounts, had improved the tin-

nitus in two of his cases. He had recommended Scotch whiskey with luncheon and dinner, and in a few weeks the tinnitus had entirely disappeared. In another case, in which the man was overworked, he prescribed a little port with meals, with resulting improvement. Some individuals needed to eat less and others more, and consequently the habits of patients in this regard should be ascertained.

Dr. Sheppard, in closing the discussion, reiterated that while importance attaches to what one eats and how one eats, the question of elimination was an equally important one in the entire subject under discussion. He had seen less injury resulting from smoking among women than any other factor. Referring to the matter of the age of these patients, he had seen the same conditions in young children. Carbohydrates were more often the cause of trouble with children than with adults, this being particularly noticeable among the poorer classes who eat inferior grades of candies, etc. He had made no observations with reference to the use of salt. He had seen cases of tinnitus improved by the moderate use of alcohol. He recalled one patient, a woman, who always felt better with regard to tinnitus after a champagne dinner. He considered this a question of blood pressure.

TWO CASES OF SARCOMA OF THE DURA MATER ARISING IN THE VICINITY OF THE MASTOID PROCESS, WITH VAGUE SYMPTOMS SIMULATING MASTOIDITIS. OPERATION IN EACH CASE FOLLOWED ULTIMATELY BY DEATH.

DR. JOHN F. BARNHILL, Indianapolis, Indiana.

Mrs. C. F. B., age 36, a woman of good build, consulted me at my office June 24, 1912, concerning a pain in the right ear and the right side of the face. She stated that she had suffered some pain in the region of the ear for ten years, and that two years ago the pain was intense in the ear for a short time; that there was then a slight aural discharge, which soon ceased, after which the pain was relieved, but that there was still a feeling of soreness and occasional sharp pain deeply situated in the ear, but not behind the ear or over the mastoid. Three or four months ago the pain in the ear and over the face gradually increased and was always worse at night, preventing sleep during the morning hours. The character of the suffering frequently seemed neuralgic, starting in the face and radiating toward and into the ear. She could recall no injury to the face, ear or mastoid region. She had never suffered from dizziness, nausea or general headache.

On objective examination it was observed that the patient was anæmic, and looked worn and debilitated from the long continued suffering. Strabismus of the right eye was present, due, it was stated, to a convulsion which took place two years previously. No pupillary disturbance. Slight tenderness over the right side of the face, and on deep pressure over the mastoid. Hearing almost absent in right ear, having been lost, according to the patient, about ten years ago. Bone conduction absent. The appearance of drum membrane was almost normal.

Appearance of nostrils quite normal. Good nasal breathing. No evidence of pus in either nostril or of any sinus

disease on either side. Pharynx and nasopharynx normal. Tonsils moderately enlarged with deep crypts containing caseous material. The teeth contained many fillings, but none were openly decayed or sensitive. There was no rise in temperature or increase in pulse rate. As a result of this examination I could make out no definite ear disease, but was satisfied that the cause of the pain was not in any of the accessory sinuses. The presence of enlarged and infected tonsils, and of the many fillings in the teeth suggested more than any other condition the possibility of origin in these structures. I emptied and disinfected the tonsil pockets and advised that the patient consult a qualified dentist with the view of ascertaining positively whether or not the pain arose in the teeth.

I heard nothing further from the patient until August 1, 1912, when it was stated in a letter that Mrs. B. had continued to suffer without intermission, the former pains being now quite unbearable. She had recently consulted Dr. Albert E. Bulson, a thoroughly competent otologist, who, after careful physical and laboratory tests, had, according to the letter, stated to her that a mastoid abscess was present and that an immediate operation was imperative. I next saw the patient August 6, 1912, at which time there was nothing new worthy of record further than the continued evidence of persistent acute suffering. I was unable after this second examination to determine with any degree of certainty the cause of her trouble and so stated. It seemed possible that a latent and anomalous mastoiditis was present, but such a diagnosis could be based only on experience with other somewhat similar cases of mastoid anomaly, and not on facts gained from the present examination. The patient had, however, fully resolved to have any operation performed that promised relief, and was even willing to undergo exploratory operative measures. Before undertaking surgical measures, I advised a complete medical examination, and Dr. Charles P. Emerson accordingly went thoroughly into the case on the same day. In addition to my own findings, Dr. Emerson ascertained that there was no general cause for the head pain,

that there was leucocytosis (14,000), and that there was slight paresis of the seventh nerve. He further states that "some features of the case would lead one to suspect a central meningeal trouble involving the sheaths of the seventh and fifth nerves and secondarily involving the mastoid." And further in the report adds, "we feel that the mastoid on the right side would better be explored."

August 7, 1912, an exploratory mastoid operation was performed. More extensive incisions than usual were made and the bone of the mastoid region was widely exposed by reflecting the tissue flaps thus outlined. The bone looked softened and at one point about an inch posterior to the external meatus, granulation buds were beginning to protrude through to the outer surface. On chiseling away the surrounding osseous structure a large granulating mass was encountered which occupied the entire cellular environment backward, inward and upward. When well uncovered the attachment of this mass was found to be on the dura over and posterior to the sigmoid sinus, and over the posterior lower portion of the temperosphenoidal lobe. In removing it rather severe hemorrhage resulted, from the tumor itself, but chiefly from the sigmoid sinus, the wall of which was perforated in the process of separation.

As a result of the operation the patient was greatly but not entirely relieved from the former pain, the wound healed rapidly and closed completely within a month, enabling the patient to return to her home, some considerable distance. Within a few weeks the pain began to increase in severity, strength and mentality gradually failed, and death occurred March 1, 1913, as a result of some "brain trouble." I never saw the patient after October 1, 1912, but it was reported to me that there was no external evidence of the return of the growth.

Laboratory examination of the specimen removed showed Endothelial Sarcoma.

Pathologic Report on Specimen Removed from Case of Mrs. C. F. B., by A. C. Shipp, Path. Dept., Ind. Unv. Sch. of Medicine.

The section sent me for diagnosis shows masses of endothelial cells arranged in irregularly shaped concentric whorls about atypically formed blood-vessels. The cells vary in shape from that of a typical endothelial cell to almost spindle shape in some whorls, while in others the cells are in appearance and arrangement very similar to the cells of the round celled sarcoma. There is a slight connective tissue stroma connecting the whorls and the entire growth is very vascular. The picture as a whole is that of a malignant neoplasm, which for want of a better name we call "endothelial sarcoma."

In explanation of the above we will say that according to Mallory, "An endothelioma is a tumor of mesenchymal origin of which the cells tend to differentiate into flat endothelial cells, and to line vessels, cavities and surfaces." Of like origin is the sarcoma. The endothelioma, according to Adami, are transitional growths, (Adami & McCrea, Page 227), and their cells revert readily to the sarcomatous type. The same author gives the following description of the dural endothelioma.

"Endotheliomas are firm, flattened, or nodular tumors, originating from endothelias cells covering the dura, or, possibly from the lining membrane of the subdural space. The growth readily implicates the pia-arachnoid, and may finally extend to the brain, which it compresses or invades. Irritation of some kind seems to be a potent factor in the causation. We have twice seen post mortem, cases in which a spur of bone projecting from the inner surface of the parietal bone formed the center about which an endothelioma developed. Occasionally, these tumors spring from the outer surface of the dura and erode the calvarium, finally appearing externally. Microscopically, they present the appearance of rich branching and anastomosing bands of flattened cells, tending to be spindle shaped, with a characteristic concentric ar-

rangement. It is not infrequent to meet with tumors showing histological transitions from the endotheliomatous to the sarcomatous type (See Vol. 1, page 824)."

It is such a transition as above mentioned that is shown so plainly in the histological picture of your section, that regardless of the different opinions as to diagnosis, I still feel that this is a tumor such as is described as an endothelial sarcoma.

Case II. Sarcoma springing from the dura of the mastoid region. (Fig. 1.)

September 5, 1909, I was called to see R. S. of Centralia, Missouri, on account of mastoid disease. I found patient in bed and much prostrated. He was 28 years old, tall, well proportioned and of athletic appearance. He gave a history of pain in the ear and mastoid some weeks previously, the pain having grown steadily worse until surgical means of relief were sought and a simple mastoid operation was performed in the University hospital at Columbia, Missouri. The mastoid wound had never completely healed, the pain continued to grow worse, and weakness of the patient increased to the date of my visit. I found the mastoid wound dressed and the head bandaged in the usual way. On removal of this dressing a large, soft mass protruded from the mastoid wound which, judged by appearance, was a hernia of the brain. There was much infiltration of the skin and other soft structures around the protruding mass, the adjacent cervical glands were moderately enlarged and tender, and thin pus bathed both the tumor and its environment. The patient had always regarded himself as an exceptionally strong man, had never before been ill, and could recall no injury to the mastoid or ear, which latter had never suppurated. Pulse and temperature were only slightly disturbed, there was no marked headache and no vomiting or other symptom of intracranial pressure. I secured a specimen of the growth and the laboratory report was that it was undoubted sarcoma.

The patient was brought to the Methodist hospital, Indianapolis, where I operated September 8, 1909, removing the

growth, adjacent bone and soft structures to what appeared to be healthy tissues on all sides. Although removed thus

R. S.

Sept. 8, 1909.—Operation for removal of a large fleshy mass growing from mastoid wound.

widely there was evidence of return of the tumor with the first repair granulations, and within two weeks the mass was

large enough to protrude beyond the skin level. Death took place from meningitis three weeks after the last operation.

The base of the growth was firmly attached to the dura with which it was so intimately amalgamated that no line of demarkation could be seen. Considerable hemorrhage resulted from the separation of the tumor but fortunately the sinus was not torn open in the dissection.

In a partial search of recent otological literature I find only one other very similar case reported, that of Dr. Otto Glogau, of New York. (Annals of Otology, Rhinology and Laryngology, June, 1911). In case one, the history of which was quite complete, there was never any positive evidence previous to the operation, which in any way indicated the exact nature of the diseased process. There was not in my opinion any positive evidence of mastoiditis or even of mastoid involvement of any kind. The symptoms were very similiar, however, to cases of anomalous mastoiditis I have several times seen and operated, and it was my expectation that this case would be of that type.

I was unable to get a complete history of the second case but believe from an analysis of the statements by the wife that the early symptoms pointed more directly to the mastoid and that it was early diagnosed as mastoiditis without aural discharge.

Report of a Case of Sinus Thrombosis with Extensive Operation on the Sinus and Jugular Vein, in which Symptoms of Cavernous Sinus Thrombosis Later Developed but with Ultimate Death.

I was called to the home of Mr. B., age 55, on account of persistent, severe headache, November 10, 1913. The family physician stated that this complaint had been made for several weeks, but that the headache had grown much worse in the past two weeks, and for the past few nights had been so severe as to require the hypodermatic use of morphia. He further stated that he found on the most careful examina-

tion no good cause for the pain, but suspected that a chronic though slightly discharging ear might be responsible. Patient gave a history of moderately good health all his life. Right ear had discharged for more than forty years but he had never in any way suffered from it. He had never had any treatment for the ear and did not know the cause of onset. During the past three months has had occasional pains in right side of the head but only in the past two weeks had he felt the need of the services of a physician. The pain was more severe over the temple than elsewhere, and never centered in or around the ear; was worse at night. The discharge from the ear had never been profuse, and he had scarcely thought of it lately. Had had no vomiting, was not much dizzy and no falling or whirling sensations. Did not think he had had any chill during present illness. On physical examination patient appeared very ill. Sat erect with difficulty on account of extreme weakness. Temperature 100, pulse 95. No paralysis; no stiffness, swelling or soreness of the head or neck. Pupils normal and reacted equally to light. No tenderness to pressure over the head but slight tenderness to deep pressure over mastoid. The external auditory meatus contained a small amount of partly dried, foul smelling pus. Drum membrane wanting in posterior superior quadrant. No granulations, no cholesteatoma. These symptoms taken together with the fact that no general disease could be discovered to account for the present illness seemed to justify the diagnosis of chronic mastoiditis with acute exacerbation and probable intracranial complication and I therefore advised that he be brought to the hospital for a radical mastoid operation. This advice was followed and the classical radical operation was performed November 11, 1913. The mastoid was eburnated, the mastoid antrum was small, and there were no pneumatic cells in the mastoid process. A small amount of pus was found both in the antrum and tympanum. No necrosis or fistula of the inner table was anywhere found. Following this operation the patient's condition continued same as before, and a few days of careful observation by the nurse showed several oc-

casions when there were chilly sensations but no distinct chill. The temperature chart, pointed rather conclusively to infection from some source.

Blood count was made and found to be as follows:

White blood count 23,900

Red blood count 3,800,000

Polmorphonuclear leucocytes 91%.

Small round celled lymphocytes 5%.

Large 4%.

Never any symptoms of marked intracranial pressure. Still no general disease was found to account for the trouble. At the end of a week, after the mastoid operation, the symptoms, while not wholly typical of sinus infection, we believed to originate in that vessel, and a sinus operation was therefore advised and performed. When extensively opened it was found that the sigmoid sinus contained no trace of blood but was filled with liquid pus in the lateral sinus and almost to the torcular. The bulbar end was therefore filled with a strip of iodoform gauze and the jugular vein was exposed and was found full of pus at the upper end but contained infected but not broken down clots toward the clavicle. The jugular vein was ligated below near the clavicle and above close to its exit from the skull. The branches were followed up and ligated and the intervening vein removed. The bulb end of the sigmoid sinus was then carefully cureted and an iodoform wick was inserted deeply toward the bulb. The bulb itself was not uncovered or cureted. The neck wound was completely closed except for the exit of a large cigarette drain. The mastoid and sinus wounds were packed and left widely open. The patient's condition at the end of the operation while not critical, was also not good.

Following this operation the pain was lessened and the temperature curve followed the normal line very closely for about one week, after which, it again became somewhat irregular, but did not until the 10th day rise above 100. From the 20th to the 30th there was again septic temperature. The appetite was never satisfactory, sleep was

sometimes impossible and there was occasionally a sort of muttering delirium lasting only for short periods. Patient sat up at the end of ten days and desired much to return to his home. At this period there appeared, with great suddenness, a swelling and protrusion of the right eye (Fig 2.)

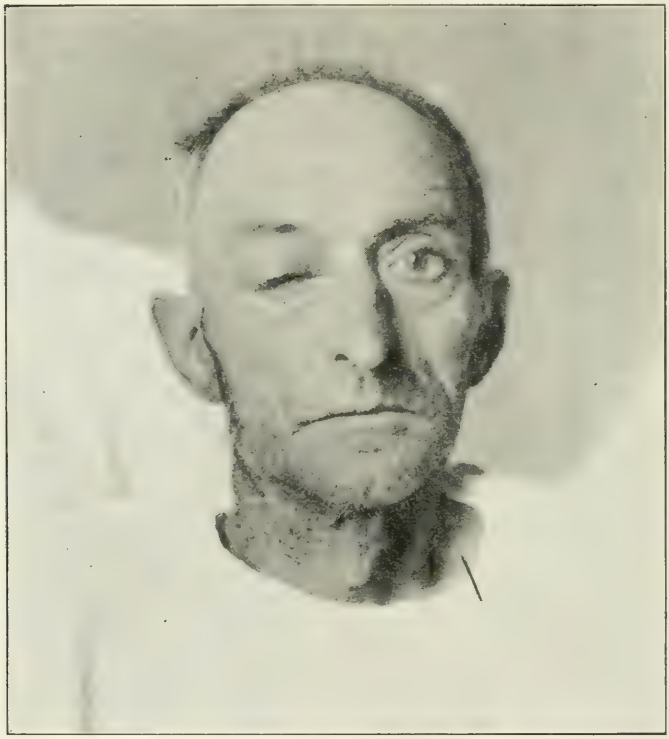


Figure II.

Lateral Sinis Thrombosis, operation, followed by thrombosis of cavernous sinus.

Separation of the lids revealed no inflammation of the eye, and vision was normal. Eye grounds were not examined. All symptoms pointed to thrombosis of the cavernous sinus, and although the progress of the case had seemed favorable till now, when the appearance of this new complication gave cause for an unfavorable prognosis. On this account, and on

account of the earnest pleading of the patient to go home he was allowed to do so, accompanied by the same nurse who had been in constant attendance. At this time the wound in the neck was completely healed and the mastoid and sinus wounds were nearly dry, healthy in appearance and almost closed. I saw the patient no more, but received frequent letters from the attending physician, saying that the progress of the case had become favorable, the temperature gradually returning to normal, the protruding swollen right eye having returned almost to normal at the end of a month, and that the strength and general well being of the patient were apparent. On January 15, 1914, recovery was so far advanced that the nurse was dismissed. The patient was then able to sit at table with his family at mealtime and on nice days had walked some in his farmyard. Death occurred suddenly on February 24th at this time of expectant recovery, the cause probably being an embolism. No post mortem was made, consequently neither the exact cause of death nor the state of the obliterated sinuses could be known, but it was believed by all who saw and studied this case that all the larger cranial sinuses on the right side of the head were at one time blocked from the general circulation.

The chief lesson in the third case is found in the fact that a running ear may continue a very long time without apparent harm, but that suppuration, though slow, is almost certain in all cases to end in disaster to the patient.

Discussion.

Dr. Stephen H. Lutz, Brooklyn, N. Y., had reported, in 1899, a case of sarcoma of the mastoid in a child nine years of age. The history of the case was published in the report of the Manhattan Eye and Ear Hospital. He had now under observation a case of endothelioma in a man sixty-seven years of age. The man had had a radical frontal and antrum operation, and the endotheliomatous tissue now protrudes from the antrum. The man was having trouble with his ear, but refused to submit to another operation. In looking up

the literature in 1899 he had found reports of thirty-eight cases of sarcoma in the region of the mastoid.

Dr. William Sohier Bryant, New York, cited a case of endothelioma of the ear reported by him two years ago. The patient was operated upon, and died afterward from mediastrual melostases.

Dr. Ewing W. Day, Pittsburg, Pa., asked if there was much pressure when the bone was removed. Dr. Barnhill answered in the affirmative. The speaker then asked if there were any signs of incoordination, to which the reader of the paper replied that there was none whatever. Without gross cerebellar symptoms Dr. Day could not understand how there could be much pressure. He had been surprised to see how many cases of extra-duval abscesses over the cerebellar give incoordination tests. He was inclined to doubt the diagnosis in the cavernous sinus case, as he could not understand how there could be thrombosis of one sinus without extension over to the other. He had been struck by the fact, as shown at autopsy, that other things will give protrusion of the eye-ball. He cited one case of injury in which the boy had infection, with protrusion of both eyes. The sinuses were found, at autopsy, to be completely obliterated. The brain pressure was so great that it had absolutely obliterated the cavernous sinuses. There was a damming back, and a manifestation of the same symptoms found in cases of thrombosis of the cavernous sinus. In another case he did a decompression operation, split the dura, and found a rupture from what he thought was the third ventricle. There was a complete subsidence of the symptoms, with permanent drainage for three months. Recovery followed, with a flow of cerebral fluid and complete disappearance of the exophthalmos. If one would bear in mind the relationship of the third ventricle to the structures in front one could readily see how protrusion of the eye-ball might be caused in such a case, but he could not understand how there could be thrombosis of one cavernous sinus and not of the other.

Dr. S. MacCuen Smith, Philadelphia, said all the cases of this kind which he had had had died promptly, and he was not able to secure post-mortem examinations. Unless confirmed by autopsy, he thought the diagnosis must remain doubtful. He referred to one of his cases at the Germantown Hospital, which was also seen by Dr. Dench, in which Dr. de Schweinitz had made the diagnosis of thrombosis of the cavernous sinus from the eye examination.

Dr. Eugene A. Crockett, Boston, Mass., was interested in the endothelioma cases, which are much more common than Dr. Barnhill seemed to think. He cited a case of sarcoma of the dura in which he had operated before the American Medical Association when it met in Boston in 1899. Another case, of round-celled sarcoma, which had been operated upon by Dr. George L. Tobey, was seen by him a month after the operation, at which time there was a fungating mass over the mastoid region. Coley's fluid was used with disappearance of the mass. Four years ago he had treated another case of sarcoma with Coley's fluid. The sarcoma disappeared, the glands of the neck subsided, and the patient was well six months ago. Coley's fluid undoubtedly effected a cure in these cases. In another case of sarcoma, with exophthalmos and all the usual symptoms, twenty milligrams of radium, buried in the orbital fat, caused absolute disappearance of the growth and of the symptoms. The treatment was begun last of September, and there was now absolutely nothing left, with the exception of a small linear scar, such as one finds after the Killian operation.

Dr. E. B. Dench, New York, referring to the pain over the front of the face, as reported in Dr. Barnhill's cases, mentioned a case with specific disease, operated upon by him, in which the pain was in this region. Of three cases of sarcoma, two in adults and one in a child, two died and one recovered. In cases of thrombosis of the cavernous sinus he would employ the Ballance operation, which he described. He had had a case with involvement of both sides, in which, of course, it was hopeless to operate.

Dr. William C. Braislin, Brooklyn, N. Y., referred to a case of sarcoma of the tonsil successfully treated with Coley's fluid. Microscopic examination confirmed the diagnosis in this case.

Dr. Barnhill, closing the discussion, said the case of sinus thrombosis presented the typical picture of what is usually seen in these cases. He gave a very unfavorable prognosis, and expressed the belief that the man would live only a few days. He was surprised when a letter came in a few days saying he was better. He was surprised to learn from Dr. Lutz and other speakers that there had been reported so many cases of endothelioma. Referring to Dr. Dench's suggestion regarding the Ballance method of getting into the sinus, it would have been very difficult in this case. It was not difficult to lift up the brain and remove or expose the gasserian ganglion. He had recently removed it, but in that case there was no pressure. In the case under discussion the congestion of the part was so great that it would have been impossible to get a half square inch exposure without protrusion of the brain substance.

Dr. Day added that if the ventricles were tapped first and everything drawn off it would be possible to get a good space, but meningitis would follow in three or four days.

FURTHER REMARKS ON THE USE OF NITRATE OF SILVER APPLIED WITHIN THE MOUTH OF THE EUSTACHIAN TUBE FOR THE RELIEF OF TINNITUS.

By WILLIAM C. BRAISLIN, M. D., Brooklyn, N. Y.

The following notes aim to elaborate certain points of a paper read before this society two years ago on the use of silver nitrate applied within the Eustachian tube for the relief of tinnitus, especially as to technique and results.

Technique: Though medicaments have been applied to the interior of the tube by the syringe, bougies and other methods, the manner of application found by the writer to be the simplest and most generally useful, is by a cotton-tipped wire. The wire employed is composed of two thin silver strands twisted firmly into one. This wire gives the proper strength, resilience and elasticity needed for the purpose, and was suggested, I believe, by Dr. Sidney Yankauer, and made by Tiemann.

The small bit of cotton for carrying the medication is wound on one end of the wire firmly and smoothly. In winding on, the bit of cotton is laid against it, and the wire rolled toward the right, as in driving a screw. The wire is rolled in the same direction when within the Eustachian tube, when necessary to facilitate its passage, for surmounting folds, or ridges of mucous membrane within the tube, if such are met with. The size of the cotton pledget when wound on the end of the wire is small, that is, rarely more than double the diameter of the wire; but in cases of marked swelling at and about the mouth of the Eustachian tube, larger pledgets are used for application within the trumpet-like expansion of the tube only. The deeper applications of the small-sized pledgets are reserved for subsequent applications. The fibres of cotton

projecting beyond the end of the wire are bent over to mark the end of the wire, and are then trimmed off smoothly with scissors in order to prevent the projecting fibres, unsupported by the wire, from doubling within the tube. The cotton pledget is then tested to see that it cannot be pulled off the wire, but is not dipped into the solution until after inflation of the tube, and then just before the wire is introduced into the catheter.

The strength of silver nitrate solution employed is usually twenty (20) grains to the ounce (four per centum) though a strength of eighty (80) grains to the ounce has been used when the application is not to be repeated within a week or longer.

Removal of Pledget from Wire After Use: If the wisp of cotton is moulded on the wire as firmly as it should be, it cannot be pulled off, and is only with difficulty, and with damage to the wire, trimmed off with scissors curved on the flat. It is most easily removed from the wire by burning off the pledget in the alcohol flame.

Under this treatment the wires may become bent or brittle. Bends and angles are readily removed by passing the length of the wire held at a slight tension, slowly through the flame. As soon as a wire shows signs of becoming brittle, it is discarded.

Results: I need hardly say that we may expect to obtain no relief from tinnitus due to lesions of the auditory nerve or of the central nervous system by this method. Congestion and swelling of the tubal mucous membrane, so generally accompanied by tinnitus, seem to me to be more rapidly relieved when in addition to the other generally employed measures, local applications of silver are made. Operative measures increasing the patency of the posterior part of the nose, sometimes alone, give prompt and permanent relief of tinnitus, especially the removal of spurs causing pressure on the opposed turbinates, posterior hypertrophy of the lower turbinates, and tumescenses from any direction causing occlusion of the trumpet opening of the tube.

Digestive, circulatory and general diseases, if present, are factors which nearly always play a part in producing or aggravating tinnitus; yet the middle ear disease once started, no matter by whatever agency, requires as a rule, active, even vigorous, treatment directed toward restoring the normal patency of the Eustachian tube.

Discussion.

Dr. E. B. Dench, New York, suggested, as a substitute for silver wire, a piece of piano wire bent sharply upon itself. This formed a little hook over which cotton could be wrapped. It was a little firmer than the silver wire.

REMOVAL OF STAPES FOR THE RELIEF OF SEVERE AUDITORY VERTIGO.

By EUGENE A. CROCKETT, M. D., Boston, Mass.

In a paper read in 1903, before the American Laryngological, Rhinological and Otological Society, I reported three cases of removal of the stapes for the relief of auditory vertigo.

At the meeting of the American Otological Society in June, 1908, a fourth case was reported, and at the Cleveland Academy of Medicine in 1912, I reported a fifth case.

It seems to me an account of the after history of these cases would be interesting. I have been unable to hear from one of the five cases, but the others I have had more or less frequent communication with and have heard from all of them within the present year.

The first case was done in 1894, and died about three years ago. His vertigo, prior to the operation, was of the most severe type. Three or four times he had fallen in the street, once he received a scalp wound from falling over backward on the asphalt sidewalk, once he fell out of his chair in his office and lay on the floor vomiting for two hours before he could recover himself. He had for several years persistent vertigo prior to the operation. For long periods he would have more or less continuous slight vertigo, then he would have periods of three or four weeks free from trouble, and then attacks of an apoplectiform variety. His ear drums were thick and retracted with some calcification, but in the left ear his hearing was practically normal.

His hearing tests were left ear, watch 37/60, voice 20/25, lower and upper limits normal.

Right ear, watch 4/60, voice 2/25, lower limit 128.

Both ears improved after catherization, but the right ear only slightly. He had the usual middle ear treatment

for over two years without any improvement to his vertigo, and at the end of that time the incus was taken out, with no improvement, and finally, at a second operation, the stapes was removed. A particularly interesting thing was that his stapes was perfectly freely movable. Dr. Philip Hammond, who assisted me, I think may remember this incident. I had been led to think that the stapes might have been fixed on account of the hearing tests. He never had any severe vertigo after the operation up to the time of his death, a period of over twelve years, but he had a slight vertigo for two or three months. His deafness was apparently total for all tests twenty-four hours after the operation.

The second case had had vertigo for two and one half years. Some of his attacks lasted three or four days, with severe nausea and vomiting, and occurred as frequently as once a fortnight for more than a year. Two months before operation they became continuous, so that he was unable to work, and he always had some tinnitus, which usually increased with attacks. Both his ear drums were very much retracted with cicatrices. Catheterizing showed open Eustachian tubes on both sides.

Hearing right ear, watch 60/60, voice 19/25, lower and upper limits normal.

Hearing left ear, watch 2/60, voice 2/25, 512, tuning fork, air connection 8″, bone conduction 15″. Normal hearing for this fork, air conduction 75″, bone conduction 25″. Lower limit 512, upper limit normal.

He had middle ear treatment for a long time without improvement. In 1902 the stapes was removed. He had not been to work on account of vertigo for over two months prior to the operation.

This patient returned to work two weeks after the operation and has had no vertigo since—twelve years. His hearing in the operated ear is at present zero for all ordinary tests.

Third case, G. T., 35 years old, was first seen Feb. 2, 1907. He was a perfectly strong, healthy man, rather neurotic and

a good deal overworked, but without any localized manifestations to account for his symptoms.

Two or three years before beginning treatment, he had noticed ringing in both ears, and for the last year he had not been able to hear well and the deafness had steadily increased. Both drum membranes were very thick, but without cicatrix or calcification; there were also marked hypertrophic rhinitis. His hearing in the right ear was normal, although the drum was thickened. In the left ear he heard a loud ticking watch at two inches, low spoken voice was not heard at all, loud spoken voice at six inches, probably by bone conduction. His lower tone limit was 512 v. s.—the high tone limit was normal.

Between two or three years before his first visit he had sudden attacks of vertigo, nausea, and vomiting lasting about half a day. For some months prior to these attacks he had had a constant feeling of pressure and lightness in his head. Subsequently he had attacks of vertigo once or twice a month from 1905 to 1907. These attacks were always precipitated by sudden change of position; as, for instance, bending forward suddenly to lace his boots, or throwing himself back in bed. During the whole of these two years he had no treatment for his ears at all, but had been to a specialist on internal medicine, who had treated him for chronic gastritis. He had been on a rigid diet for over a year without any improvement in his vertigo, in fact his condition grew worse. The general character of his vertigo was always the same. On the day of the attack he would have a confused sensation in his head, often for two or three days prior to the onset; then, as I said before, from any sudden change of position an attack would at once begin with violent vertigo of the type where the ground would seem to come up and strike him, and he would fall forward if he were unable to go to a chair or be held by some one. He would then, in two or three minutes, begin vomiting violently, and this would last two or three hours, when the attack would pass off, leaving him perfectly well for two or three weeks. The attacks

seemed to bear no relation to what he ate, and there was no loss of consciousness in any of them.

In March, 1907, the character of his attacks changed to a practically continuous vertigo. He was seldom free from a certain amount of equilibrium disturbance during the day, and he often staggered when he walked and he was afraid of being arrested for drunkenness. There was a continual sensation of fullness in his head, and a good deal of mental depression. The attacks of nausea and vomiting at the last, just before I operated upon him, often occurred once, twice, or three times a day.

An examination by a neurologist revealed no disturbance of the nervous system, the reflexes were normal, and there was no nystagmus. No amount of argument could persuade the patient that he was a subject of a curable disease. He began to be melancholic and was entirely unfit to carry on his business.

An examination of his eyes by an oculist showed no muscle trouble or refractive error beyond a slight myopia of, which had been corrected by glasses. Thinking that overwork and business worry might be a factor in his case, I advised a vacation, but after being away about ten days, during which time the patient had daily attacks of vertigo, he came home saying that he did not care for any further vacation under such circumstances.

After about two months' treatment with the Eustachian catheter and Politzer bag and cauterizing his nose, stopping his smoking and giving iodide of potasium in small doses, I advised him to have the stapes of the deaf ear removed, not promising him definitely that relief would be obtained, largely because I was afraid, owing to the extreme amount of ossicular fixation shown by the hearing test that I would be unable to remove the stapes without fracturing the crura. Notwithstanding, in June, 1907, he was etherized, and the usual flap incision made over the incudo stapedial joint. This was severed with a right angle knife and the incus removed. The stapes was clearly seen to be freely movable and was easily extracted with a right angle hook. Much to my

surprise, the interference with the low tone vibration in this case must have been other than in the base plate of the stapes. There was a good deal of disturbance of equilibration and some nausea for two days following this operation. The flap in the membrane tympani healed very promptly, and the patient left the hospital in three days and began his business in a week. He had slight equilibrium disturbances for the next month, but not enough to interfere with his business, to which he went every day. The long continued severe vertigo in this case made the patient very nervous. He still complains of slight vertigo at the present time (more than a year since the operation). He finds it hard to describe these attacks, but they are evidently only transitory and never noticeable to anyone but himself. They have never lasted more than two or three minutes at any time in the last six months, and have been entirely unaccompanied by nausea or vomiting. The hearing in the operative ear is practically gone; but, of course, it was far from good prior to the operation.

I had him come into my office three days ago. His hearing in the operative ear was absolutely zero for all my tuning forks and the whole of the Galton whistle, and there was also no voice hearing. He did, however, hear a middle C tuning fork twenty seconds on the bone. He told me he had absolutely no vertigo for over five years.

The fourth case I reported in Cleveland was operated on in March, 1911. He was a man about 57 years old and extremely neurotic. His vertigo dated back six years and was usually apoplectiform and lasted about one hour, occurring once in four or six years, and had rapidly increasing deafness and tinnitus. At the time I operated on him, he had assumed a phase of more or less continuous slight attacks, which form bothered him more than any other, as he was a man of leisure and his pleasure was running his automobile, and he could not steer with these attacks.

Two years before I saw him he had a lumbar puncture done once or twice with no improvement resulting. His hearing was normal in the left ear, and in the right ear it

was watch 2/80, voice 0/25 0/35. A middle register tuning fork was heard 10 seconds by air and 10 seconds by bone; his lower limit was 96 v. s. The drum membrane was normal and showed the incudo stapedial joint through the membrane. The stapes was removed from the right ear under ether, a sharp-pointed stapes hook being used. The incus was easily separated and the stapes hook passed through the crura, but at the moment of making traction he had a twitch of the facial nerve. The stapes was easily removed and some labyrinth fluid followed.

When he came out of ether he had a slight facial paralysis, which rapidly increased and became total, and remains so at the present time. The amount of improvement, as far as the vertigo is concerned, is marked, and he has said that the facial paralysis did not bother him at all as long as his equilibrium was all right. He has no attacks of vertigo since the operation. I heard from him over a week ago, and he repeated that while the facial paralysis was an annoyance, it was nothing to the vertigo, and that he was absolutely free from any trouble. The operated ear in this case is deaf to all tests.

In 1912, I removed the stapes on a man 70 years old. He had an attack of vertigo of the apoplectiform type going down stairs, and injured himself severely, and also had many slight attacks and had been unwilling to go out of the house on account of them. On account of having arteriosclerosis, I was uncertain whether the operation would relieve him, but it did, and I heard from him a year after the operation and he had no further attacks and was able to resume his work.

Those five cases represent carefully selected cases, but it is interesting to say that the result in all of them was perfect, no one of the five having any vertigo after the operation, although all had trouble prior to it. I attribute the success partly to the careful selection and partly to the fact that in all of them a total deafness resulted in the operation. Unless the resulting inflammation completely destroys the function of the labyrinth, a perfect result should not be

expected. The selection of the ear to be operated on was a simple matter in these cases from the hearing tests. No labyrinthine tests were necessary, therefore, and in fact most of the cases antedated this modern discovery, but labyrinthine tests would undoubtedly be of the utmost importance in many doubtful cases.

I have always felt that my three papers upon this subject have not met with much enthusiasm and have had very little discussion, and I have not known of the operation being done elsewhere. This is also a painful shock to one's vanity. It seems to me it is a very simple procedure for a very troublesome disease. I believe that it is just as efficacious as the very severe labyrinthine operation advised by modern surgical writers and followed by many imitators all over the world. Of course, no case should be operated on except after a thorough analysis of the symptoms, the absolute exclusion of any other disorder of the central nervous system, and a thorough antecedent middle ear treatment.

Discussion.

Dr. John F. Barnhill, Indianapolis, Ind., asked the reader of the paper, in closing the discussion, to explain more fully how he knew upon which ear to operate. It seemed to him that there was not enough difference in the ears to determine this. If an individual had tinnitus in one ear and not in the other, he would know which should be operated upon.

Dr. E. B. Dench, New York, cited a case of stapes fixation in which he operated, passing the knife in through the ligaments until he got a few drops of fluid. If one did not destroy the labyrinth the patient would probably continue to have vertigo. He had had a few cases in which complete cessation of the vertigo had been obtained by the injection of pilocarpin, two drops of a two per cent. solution, through a catheter into the middle ear. Sometimes when pilocarpin was used internally without results it would prove to be beneficial when used locally.

Dr. Crockett, in closing the discussion, said he did not go into the treatment prior to operation, but in the first three he had used a two per cent. solution of pilocarpin through the catheter. In the others the usual treatment had been employed. Of course, before the modern labyrinthine tests (the cases reported ran back prior to 1897) he simply selected for operation the ear with the poorer hearing. Both ears were affected. Nowadays, with the caloric test, etc., it would be a simple matter to tell which ear is affected.

THE TREATMENT OF ACCIDENTAL WOUNDS OF THE DURA DURING OPERATIONS UPON THE MASTOID PROCESS.

BY EDWARD BRADFORD DENCH, M. D., New York.

While wounding of the lateral sinus occasionally occurs in operations upon the mastoid, wounds of the dura in the middle cranial fossa are comparatively rare, — at least, if we are to judge from cases reported. While I have not searched the literature thoroughly, I have been fairly conversant with otological literature for the past twenty-five years, and I do not remember seeing in print any article dealing with this particular subject.

The paper in question, therefore, is based entirely upon my own personal experience, and consists of a series of cases in which, during the performance of the radical operation, unintentional entrance of the subdural space occurred.

It is impossible to say, in some of these cases, whether actual healthy dura was perforated at the time of the operation, or whether the instruments passed through a previously necrotic area. In certain of the cases it seemed to me that the dura must have been necrotic at the point of perforation. In other cases, I am certain that the dura was healthy, and that the opening of the subdural space occurred through healthy dura, uninvaded by any previous pathological process. Each case will be briefly detailed, and the conclusions drawn.

Case I. A young adult, with a history of chronic suppuration, presented at the New York Eye and Ear Infirmary, and the radical operation was advised for the relief of this condition. The antrum was small, and in attempting to enter it with a gouge, I found that the dura had been perforated. On enlarging the wound, the dura looked unhealthy, and I was inclined to believe that the dura had been necrotic at this point, although, naturally, this could not be absolutely

proven. The radical operation was completed, a narrow strip of iodoform gauze carried into the dural opening, the radical cavity was packed off with iodoform gauze, and the posterior wound sutured. This patient had no untoward symptoms, and made a complete recovery.

Case II. Male, about 20 years of age, suffered from a chronic suppurative otitis media. The radical operation was advised for the relief of this condition. On removing the external wall of the tympanic vault, the dura was perforated accidentally, on account of the fact that the middle cranial fossa lay exceedingly low in this patient. I was so confident that the external wall of the attic alone had been removed, that I passed a probe inward, expecting to find the internal bony wall of the attic, instead of which my probe entered the brain substance. This patient had never had any previous cerebral symptoms, and as the experience occurred in the earlier years of my practice, I was naturally greatly disturbed. The opening in the dura was packed off by a strip of iodoform gauze, the radical operation was completed in the ordinary manner, great care being taken to do a complete and thorough operation. The usual meatal flap was cut, and then the area of wounded dura was thoroughly exposed by the removal of the bony wall in every direction. A strip of iodoform gauze was carried just within the margins of the dural wound, and was brought out of the external auditory meatus. The radical cavity was packed with a second piece of iodoform gauze, and the posterior wound sutured. Absolutely no untoward symptoms followed. The gauze from the radical cavity was removed on the second or third day and replaced. The gauze in the dural wound was allowed to remain in position for four or five days, when it was removed and carefully replaced through the meatus. Absolutely no untoward symptoms supervened in this case. The patient made a perfect recovery, and was under observation for a number of years, after the operation. His suppuration was entirely relieved, and during the period of his convalescence, as well as subsequently, no cerebral symptoms developed.

Case III. A male, about 40 years of age, suffering from chronic middle ear suppuration, of long duration. The antrum was located high up, and was small. In attempting to enter the antrum a small, narrow gouge was used, and when the antrum had been supposedly entered, I found a probe passed without resistance for a considerable distance. In other words, I knew that the probe had entered the brain substance. The bony wall around this opening was rapidly broken down by means of the gouge, and the dura was found to look suspicious. I believe, in this case, that there had been a necrosis of the dura at this point, although I cannot be certain of this. The radical operation was completed, a small strip of iodoform gauze inserted through the dural opening, the radical cavity was packed with iodoform gauze, and a third strip of iodoform gauze was inserted between the packing entering the dural opening, and that filling the radical cavity. The ordinary meatal flap was formed, and the posterior wound sutured. This patient never had a rise of temperature, and never experienced any untoward effect. Ten days after the first operation the posterior wound was reopened, and the entire cavity was grafted by means of a Thiersch graft. The patient made an uninterrupted recovery. It is now ten years since this operation, the patient is in perfect health, and has never suffered from any cerebral symptoms.

I omit here two cases of similar conditions, found at operation, where, in one case, the dura had been accidentally opened by another operator, and a brain abscess was found later. Also, a second case, in which an eroded area of dura was found at the time of operation, leading into a brain abscess. Both of these cases have been reported in the series of cases of brain abscess, presented by me at the International Otological Congress, held in Boston, in 1912. One of these patients recovered completely, and the other died after being fairly well for a period of over three months. As these histories have all been published, I will not go into details regarding them here.

Case IV. A woman, between 30 and 40 years of age, presented herself at the New York Eye and Ear Infirmary, with symptoms of an acute mastoiditis. There were indefinite cerebral symptoms. The patient was immediately operated upon. The mastoid was found to be small, the inner table carious, and the dura was entered with the curette. I believe here that the dura was necrotic, as every precaution was taken at the time of operation, and it does not seem to me that an accidental wounding of healthy dura could have occurred in this case. The patient developed symptoms of meningitis, and in spite of operative interference, died as the result of meningeal infection.

Case V. The patient was a young man, who came to the New York Eye and Ear Infirmary, over two years ago, suffering from a chronic suppurative otitis media, for which the radical operation was advised. In this case the dura was accidentally entered. The bony margins surrounding the dural opening were removed in every direction, so as to expose a large area of dura. The dura was then opened by two incisions, about one-half inch in length, crossing each other at a right angle. Iodoform gauze was lightly packed into the dural opening after the completion of the radical operation. This patient had a slight temperature immediately after the operation; the temperature, however, never rose above 102 degrees, and gradually sank to between 99 and 100 degrees. He complained of slight headache, but of no other symptoms. The posterior wound was not completely closed, in this case, the dural packing being brought out through the posterior opening. The patient was observed carefully, and about ten days after the operation, showed, on examination by a prominent ophthalmologist of this city, a definite elevation of the optic papilla, on the operated side. I might say that the patient was also examined by another ophthalmologist, who found no elevation beyond the physiological limit. I explained to my consultant, that further operative procedure, in this case, would depend upon his decision. In other words, I had no other evidence of superficial brain abscess than which would

be afforded me by a beginning optic neuritis. The ophthalmologist saw this patient several times, and gave the absolute opinion that there was a slowly increasing swelling of the optic papilla upon the affected side. I, therefore, advised exploration of the wound, believing that I should be able to evacuate a superficial brain abscess. The patient absolutely refused operation, and was therefore compelled to leave the hospital. He disappeared from observation, but I learned subsequently from one of my hospital internes, that at least a year after the operation he was perfectly well. In other words, he made a complete recovery, and the slight optic neuritis from which he was supposed to suffer, was probably an error in diagnosis.

Case VI. A young woman, suffering from a chronic suppurative otitis media, was advised to undergo the radical operation. The middle cranial fossa was very low in this case, and on removing the floor of the middle fossa, a small spicule of bone must have penetrated the dura. The dura was perfectly healthy in this case. The area of dural exposure was enlarged, and the dural opening was also enlarged by two crossed incisions, at right-angles to each other. An iodoform gauze packing was introduced through the dural opening, and the radical cavity was packed off with iodoform gauze, in the usual way. A third strip of gauze was inserted between the gauze draining the subdural space, and that filling the radical cavity. The posterior wound was completely closed. A dressing was made on the third day after the operation, and on removing the gauze packing from the subdural space, about a quarter of a teaspoonful of softened brain tissue escaped. The patient was anaesthetized, the posterior wound opened, and this localized necrotic area in the brain substance was packed off with iodoform gauze. The radical cavity was also packed with iodoform gauze, and a third strip of gauze carried between these two packings so as to separate them. This patient made an absolutely uninterrupted recovery.

I have been led to report these cases because they seem to demonstrate that even accidental opening of the dura, at

the time of the radical operation, is not necessarily followed by severe sequelae, provided the operator is careful to preserve perfect technique during the operation, and to do a complete operation, even although necrotic dura may be found or an accidental opening of healthy dura occur in the early stages of the operative procedure.

I believe that the favorable termination, in all of these cases, was due simply to the fact that perfect asepsis was maintained during the entire operation. The wounded dura was meddled with as little as possible, and as soon as it was found that the dura had been opened, a packing was placed over this opening, so as to prevent the entrance of any infectious material during the remainder of the operation. Great care was taken in these cases to make the operation particularly thorough, and to see that no focus of infection remained in the middle ear.

From the report of the cases, it seems practically immaterial whether or not the original opening made in the dura be enlarged. In other words, my earlier cases, in which the small opening in the dura was simply occluded with a plug of iodoform gauze, recovered as completely as did the later cases, in which the dura was more freely opened by crossed incisions, at right-angles to each other.

I believe, however, that in these cases the proper plan of procedure is to first remove the overlying bone from the wounded dural area over a considerable extent, in order to expose a large area of dura. I believe then, after every trace of disease has been cleared from the tympanic cavity, that the dural opening should be enlarged by two crossed incisions. Iodoform gauze should now be rather firmly packed into this opening, with the idea of causing sufficient pressure to secure an amalgamation of the cerebral membranes immediately about the wounded area. In this way, general meningeal infection will be avoided. If an acute cerebritis occurs, — and this, of course, is a danger always to be thought of, — the infected area will be freely drained, and the danger of extension of the cerebritis will be very remote. This is the plan of

procedure which I have followed in my later cases, and it seems to be entirely satisfactory.

It will be noted that in only one instance did death follow this accident, and that was in a case of acute mastoiditis, with probable meningeal involvement before operation.

I have hesitated to present a paper of this kind, because no one appreciates more keenly than do I, the actual danger of dural wounds during the course of operations upon the tympanomastoid tract. I have presented it, however, because it seems to me that it is time some definite plan of treatment should be formulated for accidents of this kind, as they must occur to every operator of large experience. I do not wish to minimize the gravity of these accidents, but simply to present a definite plan of procedure which, in my hands, has proved most successful in combating them.

Discussion.

Dr. John B. Rae, New York, said that he considered it injudicious to talk of maintaining asepsis in a suppurating cavity. Injuries to the dura in the course of operation while not very common were at times unavoidable. In his opinion these injuries were more dangerous in the acute cases than in those chronic cases in which the tissue reactions, already in progress, constitute the best safeguard against the spread of infection.

Dr. S. MacCuen Smith, Philadelphia, thought the dura was diseased although it might not be recognized at the time. The resistance of normal dura to injury, even when one tried to open it with a fairly sharp instrument, was well known. The whole matter was one of drainage. The same thing held true with reference to the sinus. He had been following Dr. Dench's suggestion for two or three years.

Dr. Dench, in closing the discussion, said, in reply to Dr. Rae's question, that in two or three of the cases cited the dura was absolutely normal. He was just about to close the radical cavity when the fine probe which he was using entered a pin-point opening in the dura which must have been

made by a spicule of bone. In three instances he had to deal with wounds already made in perfectly healthy dura. With reference to preserving a sepsis in a dirty cavity, he said he simply covered up the opening in the dura with iodoform gauze until he had thoroughly completed the radical exenteration. He did not agree with Dr. Day with reference to pressure around the dural margins. Packing in between the layers of dura would help in amalgamating the pial layers and in shutting off the dural space.

THE ORBITAL APPROACH TO THE CAVERNOUS SINUS.

By HARRIS PEYTON MOSHER, M. D., Boston.

For a number of years, three at least, I have been on the lookout for a case of thrombosis of the cavernous sinus, feeling that the approach to the sinus through the orbit was feasible. Nine months ago, during my last service at the Massachusetts Charitable Eye and Ear Infirmary and under Dr. Crockett as chief of staff, a case presented itself. The patient was too sick to make a definite diagnosis, but it seemed probable that there was thrombosis of the left lateral sinus with extension to the cavernous. At operation, Dr. Emerson found the lateral sinus thrombosed and dealt with this condition by the usual operative measures, first tying the internal jugular vein. Two days later, no improvement following and the protrusion of the eye and the edema of the lids increasing, the case was kindly passed up to me.

The plan of the operation which I had in mind was to gain access to the cavernous sinus through the inner half of the orbital plate of the great wing of the sphenoid. The patient gave permission for the removal of the eye. The evening before the operation was spent in preparing for it by work upon the cadaver. At the operation the outer and posterior part of the orbit was exposed by the procedure of Krönlein. The globe of the eye was retracted inward. A short trial of this method proved that it was not successful in giving sufficient room, and so the eye and the contents of the orbit were removed. Then an opening was chiselled through the orbital plate of the great wing of the sphenoid bone, the brain retracted upward and the cavernous sinus supposedly located and incised. The man died in a few days without any further diagnostic signs and without extension of the process or improvement.

Then came the autopsy, which the hospital authorities and the enthusiasm of the house officer made possible. This was the first case in the hospital in which operation upon the cavernous sinus had been attempted. The main point to determine was whether or not the sinus had been opened. In the little group about the autopsy table I somehow felt very much alone. As the saw cut through the left side of the skull cap, pus dribbled down the blade, and when the brain was exposed the left hemisphere was flooded with it. Instantly the question was in the mind of everyone, "Did the operation result in a brain abscess?" On removing the brain, however, the abscess was found in the temporo-sphenoidal lobe, in the typical place, and was due to extension from the lateral sinus. This abscess had been missed in life, and so the completed autopsy showed, had the cavernous sinus. Instead of entering the cranial cavity, as I had tried to do through the orbital plate of the great wing of the sphenoid, I missed my bearings and had gone too high, and opened the roof of the orbit in front of the lesser wing of the sphenoid, and just above the cavernous sinus. I grazed the sinus, but even my loyal house officer could not demonstrate that it had been opened. The dura had been exposed and incised on the under surface of the brain, well back in the anterior fossa. Neither dura nor brain at this point was infected. On cutting into the cavernous sinus it was found to be filled with pus. Such in brief was the result of this operation which I had coveted, an operation which was not undertaken lightly nor without preparation. Humbled by the outcome of my efforts, I went back to the dissecting room to study the steps of the operation farther. My conclusions are embodied in this paper. I have tried the procedure some ten times. It still appeals to me, and my purpose is to make it appeal to you.

Anatomy. The Cavernous Sinuses placed one on each side of the body of the sphenoid bone, and extending from the inner end of the sphenoidal fissure to the apex of the petrous portion of the temporal bone are of considerable size and of very irregular form. Their cavity is traversed by numerous interlacing filaments which give rise to a structure resembling

that of cavernous tissue, and from this circumstance they derive their name. Enclosed in the outer wall of each are the

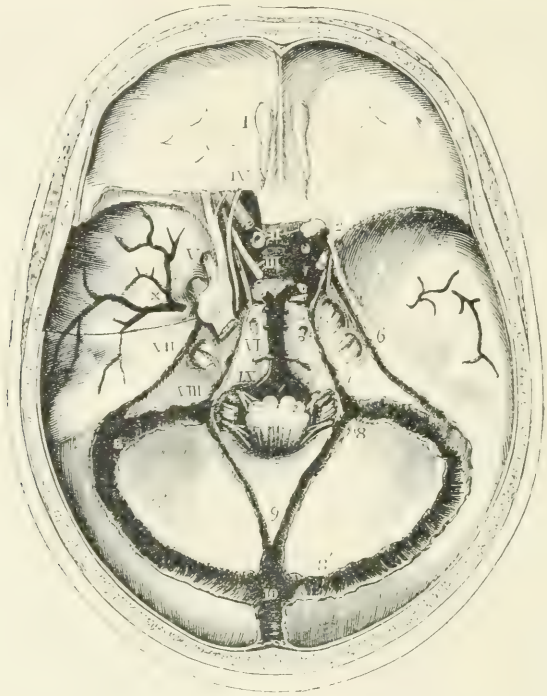


Figure 1. Internal view of the base of the skull, showing the sinuses of the dura mater (Allen Thomson). ½. The tentorium has been removed, and also a small portion of the roof of the orbit posteriorly on the left side, so as to bring into view the sinuses, which are laid open, the arteries at the base of the skull, and the trunks of the cranial nerves.

I, olfactory bulb; II, optic nerves; III, third nerve; IV, trochlear nerve; V, placed opposite to the middle of the three divisions of the fifth nerve; VI, sixth nerve; VII, facial and auditory nerves; VIII, placed opposite to the glosso-pharyngeal, pneumo-gastric and spinal accessory nerves; IX, hypoglossal nerve; 1, right internal carotid artery as it makes its turn within the cavernous sinus in the groove of the sphenoid bone; 2, its ophthalmic branch; 3, right and left posterior cerebral arteries, from the former of which the posterior communicating artery is seen passing forwards to the internal carotid; 4, basilar artery; 5, vertebral arteries giving off the anterior spinal; ×, middle meningeal artery spreading upwards from the foramen spinosum; 6, superior petrosal sinus; 7, inferior petrosal sinus; 8, termination of the lateral sinus at the jugular foramen; 8, commencement of the lateral sinus; 9, occipital sinus, in this case of large size; 10, torcular Herophili, and below that number in the figure, the superior longitudinal sinus. Quain.

third, fourth and the ophthalmic and superior maxillary divisions of the fifth nerve, as they pass forward to the

sphenoidal fissure and the foramen rotundum: and in the cavity of the sinus covered only by its thin lining membrane are the internal carotid artery, the sixth nerve, and a plexus of the sympathetic. Each sinus receives the ophthalmic veins anteriorly and communicates internally by means of the intercavernous sinuses with the corresponding vessel of the other side, while posteriorly it discharges its blood into the petrosal sinuses. It is also joined by some inferior cerebral veins, and by a small vessel named the spheno-parietal sinus (Breschet), which after receiving a branch from the dutra mater and communicating with the middle meningeal veins, runs inward on the under surface of the small wing of the sphenoid bone.

The Circular Sinus is the name given to a venous ring surrounding the pituitary body of the sella tursica and formed

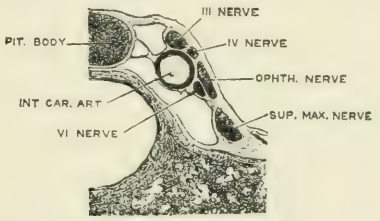


Figure 2. Transverse section of the cavernous sinus. (Langer.) Quain.

by two transverse vessels (anterior and posterior intercavernous sinuses) which connect together the right and left cavernous sinuses. Either of the transverse branches may, however, be wanting, but the anterior, which is usually the larger, is the more constant. On the other hand, there is often an additional vessel passing across below the pituitary body.

The Superior Petrosal Sinus, is a narrow canal running in the groove along the upper margin of the petrous portion of the temporal bone. Commencing at the back part of the cavernous sinus, it is directed outwards and backwards in the attached margin of the tentorium cerebelli, and ends in the lateral sinus as this turns downward in the groove on the mastoid portion of the temporal bone. It is joined by some inferior cerebral and superior cerebellar veins, as well

as by small branches from the tympanum which issue by the petro-squamous fissure.

The Inferior Petrosal Sinus which is much shorter and wider than the superior, passes from the cavernous sinus downwards and outwards in the groove between the lower margin of the petrous bone and the basillar process of the occipital bone. It passes through the anterior compartment of the jugular foramen and opens immediately into the upper end of the internal jugular vein. The inferior petrosal sinus receives some inferior cerebellar veins, and the auditory veins from the internal ear.

Variations of the Cavernous Sinus. The cavernous sinus is represented in a child by a venous plexus in the dura mater, which by enlargement and fusion of its channels is converted into the characteristic sinus of the adult. In old age the trabeculae diminish and the cavity becomes simpler.

(Quain Vol. II—Part II—pp. 523-r.)

The Sphenoidal Fissure. The sphenoidal fissure is bounded above by the lesser wing of the sphenoid, and below by the free edge of the orbital plate of the great wing of the sphenoid bone. In most cases it is a wedge-shaped fissure larger at the inner end or base of the wedge, but in some instances it is formed like an irregular dumb-bell, the inner bell being twice as large as the outer one. It is fully two centimeters long. Through it run the third, the fourth, the ophthalmic or first division of the fifth, and the ophthalmic veins. The lower margin of the fissure presents near the middle a small tubercle from which one head of the external rectus muscle of the eye takes its origin.

After the contents of the orbit have been removed and the periosteum cleaned away from the sphenoidal fissure a pair of scissors curved on the flat can be made to enter the fissure at its inner end and by hugging the lower rim it is possible to separate the dura from the outer wall of the cavernous sinus for about a centimeter. It would almost seem as if, in many cases, the cavernous sinus could be opened at this point with-

out removal of bone. For surgical purposes, however, it is necessary to enlarge the fissure. This is the essential part of the operation advocated in this paper.

The Outer Wall of the Orbit. The outer wall of the orbit is very oblique: it is formed by the orbital surface of the

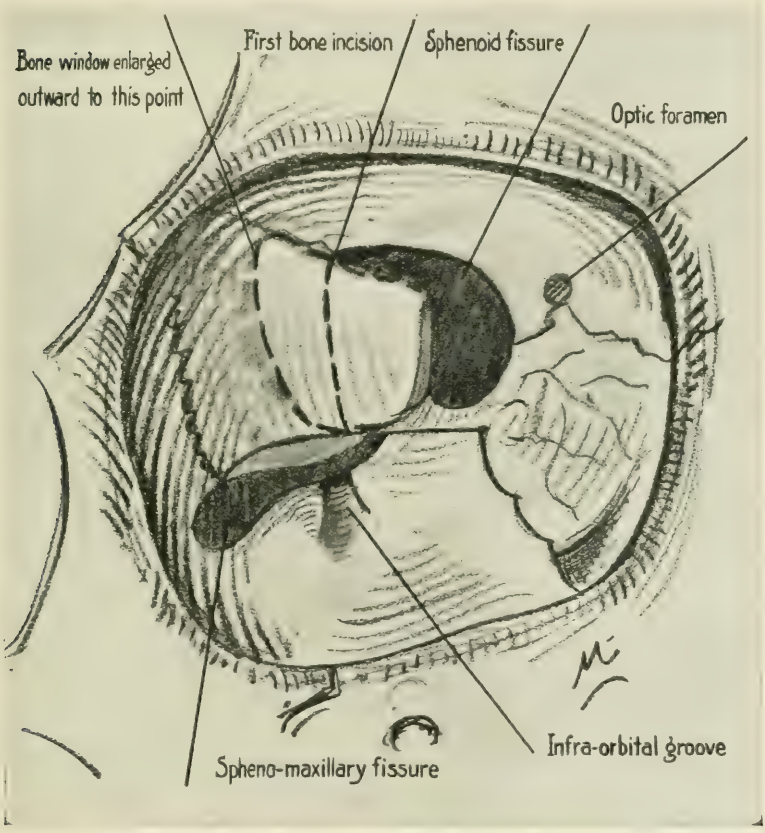


Figure 3. Drawing by the author to show the bone incisions for reaching the cavernous sinus through the orbit.

great wing of the sphenoid, and the malar. Between it and the roof near the apex is the sphenoidal fissure. Between the outer wall and the floor, near the apex, is another fissure, the spheno-maxillary, which allows the superior maxillary

nerve to enter the infra-orbital groove from the sphenomaxillary fossa. At the anterior margin of the groove the sphenoidal wing occasionally articulates with the maxilla, but frequently is excluded by the malar.

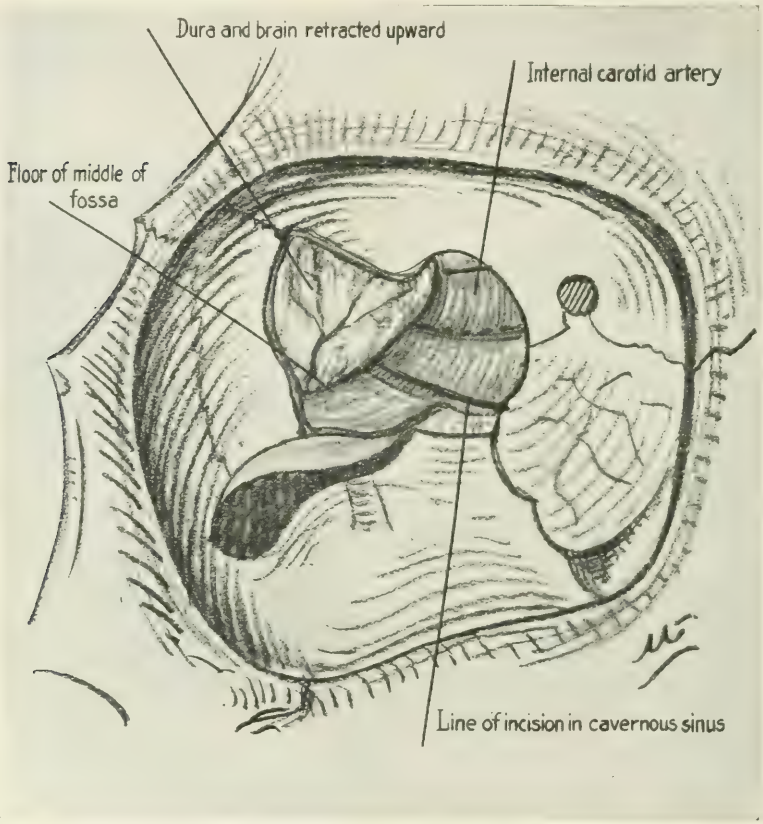


Figure 4. Drawing by the author to show the cavernous sinus exposed and incised.

The outer wall of the orbit, to recapitulate, is made from without inward, and for the first third, by the malar bone, and for the two remaining thirds by the orbital plate of the great wing of the sphenoid. Only the inner half of the orbital plate of the sphenoidal bone, however, has to do with

the cranial cavity, the anterior half, the portion which makes the middle third of the outer wall of the orbit, making a part of the anterior wall of the zygomatic fossa. We are concerned, therefore, only with the inner half of the orbital face of the sphenoidal bone, which bounds the sphenoidal fissure below, and makes the inner part of the anterior wall of the middle fossa of the skull.

Operation. The globe of the eye is removed and the orbit cleaned out. Then the ophthalmic artery is tied. The periosteum is cleaned from the posterior half of the floor of the orbit and the groove recognized in which the superior maxillary nerve runs. The next step is to separate the periosteum from the orbital surface of the great wing of the sphenoid and to recognize the outer end of the sphenoidal fissure. With the chisel placed vertically, a cut is made through the orbital plate of the great wing of the sphenoid from the notch for the superior maxillary nerve below to the outer end of the sphenoidal fissure above. The bone is thin along this line and is readily removed. It is important to make sure that the whole of the bone making the lower border of the fissure is taken away. The bone opening is enlarged outward one-half centimeter, using either rongeur or chisel. The lower edge of the bone window should be brought flush with the floor of the orbit. Unless this is done, it is not easy to get at the bottom of the middle fossa from which the dura is to be elevated. The enlarging of the bone window outward is best accomplished at least on the cadaver, by a straight chisel. The orbital plate of the sphenoid thickens quickly at the outer inferior angle, and may measure at this point one-half centimeter. If the window is carried further outward, the zygomatic fossa is opened. The bone is so thick where the orbital plate makes the apex of the zygomatic fossa, that the approach to the middle fossa and the cavernous sinus by removing first, as I did on the living, the malar bone by the method of Krönlein and then attacking the orbital plate from without inward, starts the operation on thick bone, and makes the exposure of the dura slow, consequently the operator becomes timid.

Furthermore—there are no landmarks by which to make the bone incision, so the operator may easily, as I did, go too high, and enter the anterior instead of the middle fossa. By making the bone incision as has just been described, that is from the groove of the superior maxillary nerve upward to the outer end of the sphenoidal fissure, the orbital plate of the great wing of the sphenoid splinters off in much the same fashion as the bridge comes away in a radical mastoid operation with the last stroke of the chisel. Having quickly uncovered the dura the operator goes on confidently. The bone window, made and enlarged outward and brought flush with the floor of the orbit, the dura is then elevated from the floor of the middle fossa, working from the outer boundary of the bone window inward. On the cadaver the dura can be separated from the outer wall of the cavernous sinus for a distance backward of about one centimeter, then the separation becomes hard. If the elevation is persisted in at the level of the bottom of the bone window a pin head opening is torn in the outer wall of the sinus at this point. I do not know whether this is due to dividing a vein running in the outer wall of the sinus or whether a strand of one of the nerves in the outer wall of the sinus is torn away. Above and beyond this point of adhesion between the outer wall of the cavernous sinus and the dura the two can be separated for about a centimeter further. Then the emergence of the ophthalmic division of the fifth nerve from the Gasserian ganglion halts the separation. If the attempt is made to separate the inner wall of the cavernous sinus from the outer wall of the body of the sphenoidal bone, the knife may have to be used to start the separation, but once it is started, it can be carried back easily to the limit of the sinus. Experimentation makes me feel that the exposure of the outer wall of the sinus is the better. Choosing then the exposure of the outer wall of the sinus and having separated the dura from it for a centimeter, a blunt pointed knife is placed against the sinus on a level with the floor of the orbit and the knife blade carried toward the body of the sphenoid until it is stopped by the bone. This incision opens the outer wall of the cavernous sinus

for one centimeter and is well below the internal carotid artery. Through this incision a small curette can be carried back to the opening of the superior and inferior petrosal sinuses, that is, through the whole body of the sinus. The distance from the rim of the orbit is on the average between 3 and 3¼ (in.) (7½–8½ cm.).

Given a cavernous sinus thrombosed or full of pus, what would happen in life in my opinion, is that as soon as the separation of the dura from the outer wall of the sinus was begun, the sinus would rupture. The escaping pus would then guide the curette into the cavity of the sinus.

The above operative technique is based upon one failure in the living, to which I owe much, and to which I hope some day one of you will also owe something, and to ten operations upon the cadaver. I believe that it is entirely feasible: that it is something more than a cadaver operation with which the demonstrator caps his operative course and stirs the imagination of the post graduate student. Its drawback is the necessary removal of the eye. When a reasonably sure diagnosis can be made the eye is already useless. The patient without operation is doomed. As I look at the question of the sacrifice of the eye, both scripture and surgical sense call for it.

The approach to the cavernous sinus, through the orbit, is the direct and natural way. Enlarging the sphenoidal fissure, which is the essential part of the operative procedure here advocated, is simply opening wider the anatomical gateway through which the sinus emerges from the skull.

Discussion.

Dr. John F. Barnhill, Indianapolis, Ind., thought the operation described by Dr. Mosher feasible, yet certain objections presented themselves. In the first place, the surgeon had to deal with a very sick patient, one who had probably undergone several previous operations. The chief difficulty was in lifting the brain. He asked Dr. Mosher how he held the brain out of the way in actually doing the work.

This could be done much more easily on a well person than on a patient suffering from the disease. In the latter case there would be more edema than usual, which would render the lifting of the brain more difficult.

Dr. Stephen H. Lutz, Brooklyn, N. Y., asked if the reader of the paper had employed the operation, proposed several years ago, in which entrance is through one of the nasal bones, through the ethmoid, and through the sphenoid.

Dr. Henry O. Reik, Baltimore, Md., asked what advantages this operation had over that proposed by Mr. Ballance, at the Boston meeting of the International Otological Congress, an operation which is as easy of approach and which does not sacrifice the eye.

Dr. S. MacCuen Smith, Philadelphia, asked Dr. Mosher, in closing the discussion, to go more fully into the diagnosis. He reiterated what he had said, in discussing Dr. Barnhill's paper,—that all his cases of cavernous sinus thrombosis had died. Dr. Dench had mentioned one case in which the diagnosis of thrombosis of this sinus was made, but the autopsy showed this to be incorrect.

Dr. E. B. Dench, New York, recalled the fact that Ballance had said that in the next case he would go through the orbit. Dr. Mosher had thus carried out the method suggested by Ballance. Removing the eye in these cases was justifiable.

Dr. Ewing W. Day, Pittsburg, Pa., said in all the cases of cavernous sinus thrombosis upon which he had operated both sinuses were involved with exophthalmus of both eyes.

Dr. Mosher, in closing the discussion, answering Dr. Barnhill's question with reference to lifting and holding up the brain, said it could be done. He had tried the operation mentioned by Dr. Lutz on the cadaver. It did not appeal to him as much as the one described in the paper. He had tried

the Ballance method, mentioned by Dr. Reik, also on the cadaver, but liked his own better. In making the diagnosis the edema was an important feature. Both eyes were not involved. The sacrifice of the eye was a relatively trivial matter, just as it was in malignant disease of the jaw or orbit. He did not understand how Dr. Day could tell whether both sinuses were involved. In his own case the other was not involved macroscopically. Replying to a question by Dr. Dench, regarding the cause of a sympathetic ophthalmia by the removal of one eye, he said he did not know. He had had no trouble of this kind in this connection with malignant disease.

NOTES ON THE USE OF RADIUM IN TREATMENT OF DISEASES OF THE EAR.

By W. SOHIER BRYANT, A. M., M. D., F. A. C. S., New York, N. Y.

The use of Radium in the treatment of diseases of the ear offers the means to fulfill efficiently two urgent requirements in otology: first, the stimulation of cell-growth; and second, the destruction of cells.

In the majority of chronic otological cases, the desiderata are the proliferation of normal tissue cells and the destruction of new-formed cellular and cicatricial tissues. Radio-active-therapy presents these two desirable and diverse modes of action, which can be used either singly or together. Often we wish to use both of these processes at the same time in the same ear. In hypertrophic and cicatricial states the destructive action of Radium is desired; in atrophic states the stimulating effect is sought. The destructive action of the Radium is desired, particularly in cicatricial states of the middle ear or in degenerations of the auditory nerve and labyrinthine mechanism. In atrophic and degenerative states, atrophic middle ear catarrh, otosclerosis and auditory neuritis and labyrinthitis, the action desired is stimulation of the growth of the remaining normal cells as well as the destruction of the pathological tissues.

There are four processes apparent in the action of Radium in ear diseases, and they are of varying importance: (1) the presence of the Radium capsule in the auditory canal acts as an irritant, as any foreign body acts; (2) the heat evolved by the Radium has a warming effect on the meatus; (3) the electrical activity of the Radium ionizes the surrounding matter; and (4) the radiation of the Radium produces diverse effects, depending upon the individual and upon the dosage.

The rays of radio-active substances are of three kinds. The shallow, penetrating, slow α rays, act only as a superficial destructive agent. Their action is not desirable in the treat-

ment of ear disease, and therefore they should be eliminated by filters. Another variety of rays is the rapid γ rays. These rays penetrate deeply and break up into β rays. The various intermediate rays between the α rays and the γ rays are the β rays.

These β rays are now considered the most important therapeutically. From an otological point of view also these rays are most significant, for to them are attributed the two apparently antagonistic activities of stimulation of desirable tissue on one hand and destruction of undesirable tissue on the other hand. In my treatment, I use mesothorium bromide because it is the richest in β rays.

In the use of Radium for the treatment of aural diseases, as in the use of Radium for the treatment of diseases of any kind, precise care should be exerted in every possible direction, so that there may be no over-use or no misuse. The reaction of the individual must be carefully watched and determined; the dosage of the application must be nicely gauged; and the equation of the applicator used must be carefully studied. Because of the comparative newness of the treatment there is practically no literature on the use of Radium in ear diseases.

I consider that too much care cannot be used in determining the susceptibility of the individual to the action of Radium. There is, of course, a wide range of reaction in individuals, and there is little known of the indication of the relative susceptibility of an individual; the amount of reaction to the Radium can as yet be determined only by careful experiment. We can, therefore, see the importance of the utmost caution in the employment of Radium lest we do more harm than good. Over-use shows in deterioration of the hearing. In the use of Radium, the first effort should be to avoid damaging anything. Before I applied the capsule I now use, I exposed my skin to it without harmful results for a period much longer than I have ever used it in the ear.

The technic of the radiation depends upon the kind of radioactive substance used, its concentration, upon the amount used, its area, the distance of application, the filterage, and the

length of exposure. The applications should always commence at a minimum and be gradually increased, the time and length of an exposure being governed, of course, by the result of the previous exposure. The intervals between the applications should also be carefully graded. The greatest difficulty in the appropriate dosage of Radium is the fact that the effect may be long delayed and may not appear for months after the application.

Every applicator should be considered an entirely new instrument. Each applicator has its own equation: it must be studied separately and its effect measured. Other applicators, even though containing the same amount of radio-active substance, are only a very general guide to an applicator, for the minute individual variations make a great deal of difference in the results. The applicator I use is a glass tube containing 5 milligrams of mesothorium bromide wrapped in thin tinfoil. The applications were made in the auditory canal.

The limitations or contra-indications for the use of Radium have not as yet been definitely established. Apparently the only conditions in which there is no indication for its use are those in which the functions of the ear are normal and those in which the labyrinth has been entirely destroyed, and all function annihilated: in other words, where there is nothing to use radiation for and where there is nothing to use radiation on.

Local radiation of radio-active substances does not appear to have much influence on the causal factors of ear disease, but its action offers a specific for the residual effects of ear disease; that is, radio-active therapy seems to be not so effective in the treatment of causes as it is in the treatment of the effects of the causes. The causal factor of ear disease should be eliminated before recourse is had to radiation. In order, therefore, to obtain the best and permanent results, it is usually indicated that other treatment be used and that Radium be considered an adjunct to this treatment. The practical indications for the use of radiation are all forms of functional derangement of the ear when the causal factors have been eliminated. The use of Radium in ear disease is

indicated and is most effective in static and terminal conditions. Besides these advantages, it has another inestimable value in the fact that it renders certain cases amenable to treatment that would not yield to other means.

To give a general idea of my results of the treatment with a radio-active substance, I submit the following data on a series of twenty consecutive cases. The cases are not selected on any basis other than chronicity. The cases were all seen in private practice, and all of them had been under my observation for a number of years before the radiation was used.

SUMMARY OF CASES.

20 Cases—40 Ears.

Otosclerosis	9 ears
Atrophic middle ear catarrh	12 ears
Effects of middle ear suppurations	13 ears
Non-suppurative labyrinthitis	2 ears
Hypertrophic middle ear catarrh	4 ears

RESULTS.

Hearing decreased in 1 ear	Non suppurative labyrinthitis
Hearing unchanged in 6 ears	2 atrophic middle ear catarrh
	4 effects of middle ear suppuration
Hearing improved in 33 ears	
Much improved in 14 ears	6 atrophic middle ear catarrh
	2 hypertrophic middle ear catarrh
	1 non-suppurative labyrinthitis
	3 otosclerosis
	2 effects of chronic middle ear suppuration
Restored to normal in 6 ears	2 hypertrophic middle ear catarrh
	4 atrophic middle ear catarrh

Case 1. Woman, 55 years old, very deaf. Atrophosclerosis tympanica and senile deterioration, arteriosclerosis. Loud voice heard in right ear at 6 inches; left ear at 2 inches. Politzer acoumeter not heard in either ear. Left ear radiation 50 milligram minutes; immediately afterward ordinary speaking voice heard at 26 inches. Four days later, radiation in each ear of 50 milligram minutes, after which right ear heard ordinary voice at 108 inches and left ear at 40 inches. Thirty-nine days later, after additional radiation in right ear with 100 milligram minutes, in left ear with 315 milligram

minutes and routine nasopharyngeal treatment, watch was heard in right ear 9/60 and left ear 13/60.

Case 2. Woman, 34 years old. Right ear residual condition from chronic middle ear suppuration, remains of ossicles bound in a cicatrix on posterior tympanic wall. Politzer acoumeter heard at 14 inches. Left ear heard nothing. Forty-eight days later, after radiation in right ear of 105 milligram minutes and in the left ear of 260 milligram minutes, the right ear heard Politzer acoumeter at 31 inches. With proper adjustment of a prosthesis, watch is heard 58/60, practically normal hearing. Left ear unchanged.

Case 3. Woman, 54 years old. Double radical mastoid operations had been done. Left ear practically totally deaf. Right ear Politzer acoumeter heard at 2 inches. Sixty-seven days later, after radiation in the right ear of 315 milligram minutes and 425 milligram minutes in left ear, Politzer acoumeter was heard in right ear at 13 inches; left ear at 14 inches. Patient has been transformed from an almost deaf-mute to an intelligent member of society.

Case 4. Woman, 49 years old. Left ear has had a radical mastoid operation. Right ear atrophosclerosis tympanica. Left ear heard Politzer acoumeter at 17 inches; right ear heard watch 3/60. Forty-six days later, after radiation of 100 milligram minutes in each ear, watch was heard in right ear 8.5/60 and acoumeter heard in left ear at 48 inches.

Case 5. Woman, 60 years old. Osteodistrophy petrosa. Right ear heard watch 4/60. Politzer acoumeter heard in left ear at 16 inches. Seventy-one days later, after appropriate nasopharyngeal treatment and radiation of 245 milligram minutes in right ear and 305 milligram minutes in left ear, the watch is heard by right ear 8/60. Left ear heard acoumeter at 40 inches.

Case 6. Woman, 48 years old. Osteodistrophy petrosa in both ears. Politzer acoumeter heard in right ear at 9 inches; in left ear at 4 inches. Thirteen days later, after radiation in the left ear of 135 milligram minutes and appropriate naso-

pharyngeal treatment, the right ear heard the Politzer acoumeter at 20 inches and the left ear at 11 inches.

Case 7. Man, 62 years old. Arteriosclerotic. Middle ear catarrh of the hypertrophic variety with senile deterioration and glaucosuria. Watch heard 0/60 in contact with right ear; left ear heard watch 2/60. Ten days later, after 50 milligram minutes of radiation to each ear and appropriate nasopharyngeal treatment, the watch was heard by right ear 2.5/60 and the left 3/60.

Case 8. Woman, 34 years old. Left ear middle ear destruction and exfoliation from chronic middle ear suppuration; right ear atrophosclerosis tympanica. Right ear heard watch at 0/60 on tight contact with auricle: left ear heard nothing except 213 d. v. fork. Twenty-one days later, after radiation in right ear of 115 milligram minutes, and in the left of 190 milligram minutes and routine nasopharyngeal treatment, right ear heard watch 4/60 and left ear heard Politzer acoumeter at 1 inch.

Case 9. Man, 40 years old. Effects of chronic middle ear suppuration, all ossicles and both tympanic membranes entirely gone. Watch heard 0/60 by both ears. Politzer acoumeter heard by right ear at 8 inches and left ear at 4 inches. Twenty-nine days later, after radiation of 60 milligram minutes in left ear, Politzer acoumeter was heard by left ear at 4 inches and by right ear at 17 inches.

Case 10. Man, 53 years old. Arteriosclerosis, hypertrophic catarrh and atrophosclerosis tympanica. Heard nothing in left ear. With ear trumpet heard a shout very indistinctly in right ear. Fifteen days later, after 145 milligram minutes radiation in right ear and 250 milligram minutes in left ear, right ear heard without the trumpet a loud voice at 4 inches and the left ear heard nothing.

Case 11. Woman, 20 years old. Patient had had radical mastoid operations. Watch heard in left ear 4.5/60 and in right ear 0/60 and Politzer acoumeter at 45 inches. Twenty-five days later, after radiation in right ear of 145 milligram minutes and in left ear of 190 milligram minutes.

watch was heard by left ear 10/60 and by right ear Politzer acoumeter was heard at 108 inches.

Case 12. Man, 35 years old. Hypertrophic middle ear catarrh. Watch was heard in left ear 1.12/60 and in right ear 11/60. Fifteen days later, after radiation of 15 milligram minutes in left ear, watch was heard in left ear 60/60 and by right ear 60/60.

Case 13. Man, 19 years old. Radical mastoid operation had been done in right ear. Left ear chronic middle ear suppuration with destruction of tympanic contents; tympanum full of granulations, polypi and fetid discharge. Right ear almost entirely deaf. Left ear has very poor hearing. Politzer acoumeter heard by right ear at 0 and left ear at 10 inches. Eighty-six days later, after radiation in right ear of 525 milligram minutes and in left ear of 600 milligram minutes, the left ear had ceased to discharge and was entirely healed. Politzer acoumeter was heard by left ear at 40 inches; watch 1.06/60; acoumeter in right ear 0. inches; ordinary voice heard at 3 inches.

Case 14. Man, 27 years old. Right ear atrophosclerosis tympanica. Left ear chronic middle ear suppuration, central perforation. Tinnitus. Watch heard in left ear at 4 inches; right ear at 10 inches. One hundred and five days later, after appropriate pharyngeal treatment and radiation in right ear of 445 milligram minutes and in left ear of 460 milligram minutes, the tinnitus had ceased, the discharge from the left ear quickly stopped, and the perforation contracted to the size of a pin point. The watch was heard by left ear 4/60 and by right ear 15/60.

Case 15. Man, 35 years old. Toxic labyrinthitis right ear; very deaf; left ear heard watch 22/60. Seventy-six days later, after radiation of 335 milligram minutes, watch was heard by left ear 18/60 and by right ear Politzer acoumeter was heard at 3 1/2 inches.

Case 16. Woman, 32 years old. Left middle ear destruction from suppuration. Right ear osteodistrophy petrosa and atrophosclerosis tympanica. Left ear heard nothing; right ear

heard loud voice at 2 inches. One hundred and nine days later, after radiation in right ear of 265 milligram minutes and in left ear of 305 milligram minutes, ordinary voice was heard by left ear at 6 inches. Acoumeter was heard at 2 1/2 inches by right ear.

Case 17. Woman, 40 years old. Osteodistrophy petrosa, atrophosclerosis tympanica. Politzer acoumeter heard by left ear at 6 inches and by right ear at 2 inches. Ninety days later, after radiation in right ear of 480 milligram minutes and in left ear of 565 milligram minutes, Politzer acoumeter was heard by right ear at 10 inches and by left ear at 38 inches.

Case 18. Woman, 42 years old. Osteodistrophy petrosa. Watch was heard by right ear only on firm pressure on auricle; watch was heard by left ear 4/60. Twenty-eight days later, after radiation in right ear of 25 milligram minutes and left ear of 60 milligram minutes, watch was heard by left ear 16/60 and by right ear 2.5/60.

Case 19. Woman, 58 years old. Atrophosclerosis tympanica. Watch was heard 0/60 by both ears. Politzer acoumeter was heard by right ear at 8 inches and by left ear at 42 inches. Fifty-six days later, after radiation in right ear of 215 milligram minutes and 160 milligram minutes in left ear, watch was heard by right ear 1.5/60 and by left ear 2/60.

Case 20. Man, 53 years old. Atrophosclerosis tympanica. Watch at right ear was heard 1.25/60 and by left ear 3/60. Seventy-six days later, after radiation in right ear of 245 milligram minutes and of left ear of 270 milligram minutes, watch was heard by right ear 37/60 and by left ear 26/60.

(In the following table, mm indicates milligram minutes.)

NO.	AGE.	DIAGNOSIS.	HEARING BEFORE RADIATION. A. D.	HEARING BEFORE RADIATION. A. S.	RADIATION. A. D.	RADIATION. A. S.	HEARING AFTER RADIATION. A. D.	HEARING AFTER RADIATION. A. S.
1	55	Atrophic middle ear catarrh, both	0	0	150 mm	415 mm	9/60	13/60
2	34	A. D. Effects of middle ear suppuration A. S. Atrophic middle ear catarrh	14/2400	0	105 mm	260 mm	31/2400	0
3	54	Effects of middle ear suppuration, both	2/2400	0	315 mm	425 mm	13/2400	14/2400
4	48	A. D. Effects of middle ear suppuration A. S. Atrophic middle ear catarrh	3/60	17/2400	100 mm	100 mm	8.5/60	48/2400
5	60	Otosclerosis, both	4/60	16/2400	245 mm	305 mm	8/60	40/2400
6	48	Otosclerosis, both	9/2400	4/2400		135 mm	20/2400	11/2400
7	62	Hypertrophic middle ear catarrh, both	6/2400	2/60	50 mm	50 mm	2.5/60	3/60
8	34	A. S. Effects of middle ear suppuration A. D. Atrophic middle ear catarrh	12/2400	0	115 mm	190 mm	4/60	1/2400
9	40	Effects of middle ear suppuration, both	8/2400	4/2400		60 mm	17/2400	4/2400
10	55	Atrophic middle ear catarrh, both	Hears shout with ear trumpet	0	145 mm	250 mm	Hears voice without ear trumpet	0
11	20	Effects of middle ear suppuration, both	45/2400	4.5/60	145 mm	190 mm	108/2400	10/60
12	35	Hypertrophic middle ear catarrh, both	11/60	1.12/60		15 mm	60/60	60/60
13	19	Effects of middle ear suppuration, both	0	10/2400	515 mm	600 mm	0	40/2400
14	27	A. D. Atrophic middle ear catarrh A. S. Effects of middle ear suppuration	10/60	4/60	445 mm	460 mm	156/60	4/60
15	35	Non-suppurative labyrinthitis, both	0	22/60	335 mm		3.5/2400	18/60
16	32	A. D. Otosclerosis A. S. Effects of middle ear suppuration	0	0	265 mm	305 mm	2.5/2400	0
17	40	Otosclerosis, both	2/2400	8/2400	480 mm	565 mm	10/2400	38/2400
18	42	Otosclerosis, both	6/2400	4/60	25 mm	60 mm	2.5/60	16/60
19	58	Atrophic middle ear catarrh, both	8/2400	42/2400	215 mm	160 mm	1.5/60	2/60
20	53	Atrophic middle ear catarrh, both	1.25/60	3/60	245 mm	270 mm	37/60	26/60

Discussion.

Dr. Eugene A. Crockett, Boston, Mass., had had a good deal of experience in the treatment of otosclerosis by means of radium, having conducted a series of experiments at the Cancer Hospital of Harvard University. He had employed a radio-active solution advised by Duane, of the therapeutic department, in eight cases, beginning the treatments last fall. He began with two milligrams for five minutes, then for ten minutes, gradually working up to twenty milligrams for fifteen minutes. The majority of the patients had been treated for years by various other measures. He could not say that the results were as brilliant as those described by Dr. Bryant. In only three cases was there any improvement in hearing. Tinnitus was improved only temporarily. In one case of otosclerosis in a girl of eighteen the patient had passed from practically normal hearing, with a little tinnitus, to complete otosclerosis in a little over a year. She improved under treatment with radium. He felt that radium was anything but a brilliant cure, and it was very doubtful whether any permanent results could be obtained by this method of treatment.

Dr. Ewing W. Day, Pittsburg, Pa., had inaugurated a series of experiments with radium at the request of the Standard Chemical Company, which produces the major part of the radium of the world. No attempt was made at classifying the cases because, as Dr. Bryant had said, the indications and contraindications have not been established. He used five milligrams of radium, with a lead screen. Ten treatments were given, beginning with ten minutes and lengthening the time to one-half hour, and increasing the dosage to twenty milligrams. The treatments were carried up to as high as forty applications. In two cases there was improvement in hearing. In one of these, whom he had treated from 1900 to 1905, there was more improvement than in the other. The patient first heard a clock tick, and then began to go to church, but after a month the hearing was as bad as before. She thought she could hear better.

but tests showed that she could not. All work with radium had been discontinued. It seemed to stop tinnitus for a while, but he doubted whether radium would bring any remote results.

Dr. Frederick L. Jack, Boston, Mass., could not see how any benefit could be hoped for from this treatment unless radium exerted some influence upon bone formation. In many cases the deafness was due to bony fixation of the stapes. That had been demonstrated to his satisfaction in eighty cases, (a large proportion of which he had been unable to remove the stapes). Politzer had mentioned this point in connection with his cases.

Dr. Crockett, answering a question by Dr. Bryant, as to whether he used micro-milligrams or milligrams, said Duane employed the term *milliquere*, the amount being equal to a milligram.

Dr. Bryant, in closing the discussion, said Dr. Day's remarks emphasized the point that progressing cases should not be considered suitable for radioactive treatment. In static cases, or those in which the cause of the disease can be eliminated, the effects of radioactive treatment could be measured. He did not think radium exerted any influence upon the cause of the ear trouble. Radium would affect bone if used in sufficient dosage.

VARIOUS OTITIC FACIAL PARALYSES.

By B. ALEXANDER RANDALL, M. A., M. D., Philadelphia.

The otitic causation of many cases of Facial paralysis had been clearly shown before the radical tympano-mastoid evisceration had unpleasantly accentuated the relation; and some expositions of the subject, like Putzel's article in Buck's Reference Handbook of the Medical Sciences, cover well most of the ground. Yet reiteration of so important a topic has its value, and a series of recent cases, recalling and amplifying earlier experiences, seem worthy of citation at this time. Lest Dr. Holt and others should say of me, as John Green once said of one of the members, "We have to search the dustiest archives of the Society to find the new things he brings forward," I would here acknowledge my debt to many from Wilde onward, who have contributed to its discussion.

Bell's explanation of Facial palsy as due to rheumatoid inflammation at the stylo-mastoid exit, masked for a while those cases starting in the middle ear and marked by a Neuritis extending outward to become most notable at the foramen. With manifest tympanic trouble, I have seen this picture so striking as to seem purely external and by good absorbent medication as of Ung. Iodini Comp. have gained prompt relief in apparent verification of Bell's view. Again, I shall always remember a baby of four months, whose face was partially paralyzed on the right, where his polyp-filled tympanum called for intervention. Under ether I gently probed to locate the origin of the growths, recognizing a bare upper margin and a loose spicule of bone below it, which lifted out proved to be the anterior half of the stapes. The remnants of malleus and incus were removed and the tympanum eviscerated and healing soon gained, but without influence upon the paretic Facial. Lesion above the oval window seemed assured, until we noted that the paresis varied

in exact relation to the involvement of the pre-auricular lymp-nodes, which like all those of the neighborhood underwent suppuration and evacuation to the number of thirty. Dare I admit that these glands were not excised but only curetted one by one as they showed need? though I may say that my advice of sweeping removal was over-ruled by my consultants. The patient at 18 is a hearty boy, almost unscarred, unparalyzed—though with little hearing in that ear.

A recent case presented a polyp-filled drum and slight facial involvement, and the girl of 16 rebelled at probing and biting out under cocaine of the growths. Her mother was informed of the need of this or of more radical measures, and brought her back a submissive and exemplary patient. The polyps grew almost as fast as removed, but we were gaining for a couple of weeks, when she came with almost total facial paralysis. A sequestrum could now be felt, and on removal proved to be the apex of the cochlea and part of a lower turn as a second fragment; and the paresis almost completely cleared at once. Two or three weeks later the ear was dry, and with fair hearing for tones above 200 d. v. s. A month later she has suffered some renewal of the discharge; and her unusually favorable showing may prove quite too good to be true.

A man of 42 came ten years ago with his left ear devoid of hearing from chronic suppuration and a firm polyp protruding from the meatus. The scarlike walls of the dilated external meatus were exquisitely sensitive, as such often are, but I was able to carry my snare down to the broad posterior base and cut off the growth. The freely bleeding stump was cauterized with chromic crystal, but one of the repetitions was too vigorous and was followed by severe pain and facial palsy rapidly developing to totality. This took several months to clear up and has left, probably for years, that slight lack of coincident action, which is apt to persist after such paralysis. His hearing remains useless; but the result is probably at least as good as if I had eviscerated, as very up-to-date colleagues will blame me for not having done, if he would have consented to it.

I have always taken the stand (sometimes literally as a witness for a colleague) that an expert operator may precipitate palsy by his work along the facial canal, but only in such cases as are on the verge of a spontaneous and probably persistent injury by caries about the nerve; yet this presupposes full knowledge of the anatomy and an unfailingly skilful technic, such as not all possess who essay the operation of tympano-mastoid evisceration. A case was mentioned in discussing Barnhill's contribution on the tolerance of the Facial, where we found the naked nerve spanning the walnut sized cavity formed by a large cholesteatoma packed around it. As no twitching nor paretic symptoms had warned us of its exposure, it was slightly bruised in turning out the skin masses which surrounded it. The Acoustic might also be supposed to have suffered, since with facial palsy on this side, total deafness ensued on both sides; although with less extensive evisceration on the left, the internal meatus had not been approached in either intervention. Shock or the general effect of the double operation may better be held responsible for the acoustic paresis; like the facial, it slowly recovered, as later reported by the patient in his distant home.

A less usual accident is reported among my extra-dural abscess cases, where in curetting the tegmen after evacuating the extra-dural pus and the cholesteatoma mass which protruded up from the tympanum, I may have done injury at the hiatus Fallopii. I am morally sure that I did not damage the Facial elsewhere in its course—the twitchings of the face for months growing worse up to the operation, bespoke an irritative exposure of the nerve as pre-existing.

A recent patient was a man of 45 who had suffered with the rest of his household a streptococcus tonsillar attack, and his left ear, affected on a previous occasion, had been severely painful and was almost disabled by pounding pressure. Its hearing was practically normal to all tests except speech, better than the slightly catarrhal right ear, to which Weber was lateralized; but bone-conduction was reduced, facial palsy was distinct though slight, and he

complained of dimness of sight. I examined his eye-grounds with decided apprehension of beginning pontine angle tumor; but they were as normal as his drumhead; while a crusted lesion on the antitragus was clearly enough herpetic to furnish the clue to his trouble. Recovery was prompt under tonic measures, though his mouth-angle felt stiff to him two months after all visible defect had disappeared. The Facial had not seemed involved in the few other cases of "Zone auricularis" which I have encountered—once coincident in man and wife.

The treatment of these cases often lacks full effectiveness because the traditional main dependence is upon electricity. Yet most recognize that so long as symptoms of neuritis are present it is too early for safe use even of galvanism. Yet in a young "quarter-back" who was knocked briefly unconscious and left too dazed to recall his signals, vigorous use of iodide in two weeks cleared up the hæmotympanum and facial palsy which soon followed his injury, although his hearing is still very defective in this ear for low tones rather than high. In cases brought to me in consultation months or a year after injury by operation, I have seen good recovery under alteratives, far more prompt and complete, I believe, than could have been gained by the contemplated nerve "anastomosis" which I advised against.

In this connection I wish to report a case, the most signal that has come to my knowledge. A girl of 21 with suppuration on the left all her life, came to me Christmas eve, 1907, with mastoid pain and swelling, slight vertigo but no nausea or eye-ground changes. Flow had been irregular and stringy, the hearing variable but now fair except for deep tones, and inspection showed upper posterior perforation with cholesteatoma and sagging wall. Evisceration that afternoon showed remnants of the carious ossicles and the dense, eburnated cortex which (with Buck rather than Cheatle) I believe to be the result of chronic mastoid suppuration. Next day she showed some paresis of the lower distribution of the Facial, which gradually increased in extent and degree until it was total on the left side. Pinching

massage was well borne, but with alterative tonics quite unavailing; electricity was later employed with no better success; and ten months later the neurologist reported total paralysis, degeneration reaction and commencing contraction. Healing of the middle ear was followed by occasional slight returns of suppuration as nasal colds penetrated up the unclosed eustachian tube; so she was kept under some observation and encouraged to continue her massage, if only for the prevention of contraction. Full four years passed without notable change in the facial paralysis, during which I would have perhaps considered anastomosis operation had she been ready, although later most men would have felt that it was too late: then she began to show some power in the lower face, increasing visibly under the massage; and this, supplimented by tonics, has brought her to what any "anastomosis" operator would extol as a brilliant cure. Some trace of contracture is present to make the dimple and creases of this side of the face rather deeper than on the right. The eyelids cannot be fully closed and the frontalis acts still less. Coincident motion of the two sides of the face is imperfect and has not been practiced before the mirror, as will be needed to re-educate the face; but under the circumstances the success is truly remarkable and should serve as a strong support to optimistic persistence in dealing with such cases.

As alterative and absorbent medication I am apt to begin with iodide and bromide, while inflammatory changes are probably acute and pain often calls for sedative action: later I prefer liq. arseni et hydragyri iodidi, when the arsenic promises tonic rather than irritative effect. But my main dependence is upon a pinching massage of the facial muscles. Nothing can be simpler nor easier for the patient to carry out at home; it can be begun immediately and pressed lightly many times a day, since it ought to be harmless. It will often immediately double the effect of a remnant of power, as effectively as electricity yet with little risk of exhausting the excitability of the muscles and their nerve-endings; and upon the nerve trunks it can probably

act both directly and indirectly by tropo stimulation. In a condition which is commonly a nuisance to the aural surgeon and too often an aprobium if it can be ascribed to his intervention, this simple measure constitutes perhaps our greatest aid in restoring the function of the paralyzed muscles of the face.

Discussion.

Dr. E. B. Dench, New York, had had one case of temporary facial paralysis after cauterization of granulation tissue, and another in which the nerve was injured in curetting out the cavity of an extra dural abscess. In the latter case the nerve was injured internal to the geniculate ganglion and the trouble cleared up. When the injury is external to the ganglion it is more serious.

Dr. Robert Lewis, New York, cited a case in which there was a complete facial paralysis following a mastoid operation performed where the necrosis was very extensive and where under the use of electricity the function of the nerve was restored.

Dr. Charles W. Richardson, Washington, D. C., mentioned a case in which the paralysis was so complete that he thought he had destroyed the nerve. The woman was markedly disfigured. He saw her in July, and again in September when he was astounded to see how she had improved through the use of a small battery.

Dr. T. Passmore Berens, New York, recalled a case of double microtia, in which it was impossible to find landmarks, and in which complete one-sided paralysis followed operation. The paralysis cleared up entirely after a few months of massage given by the nurse.

Dr. John F. Barnhill, Indianapolis, Ind., thought the length of time in which a return of action of the muscles depended upon the character of the injury. If the injury to the nerve

were within the canal, and the bone around it were not involved, recovery would take place within a shorter time than if the bony canal were affected. One would be justified in waiting if the vitality of the nerve could be maintained. He had found this to be the case in all his work upon dogs. The return of motion after the use of electricity was sometimes so rapid as to be astonishing.

Dr. O. A. M. McKimmie, Washington, D. C., said a recent case had impressed very strongly the idea that the mastoid operation should be done without reference to mastoid symptoms other than facial paralysis when that is present. In the case in point the house surgeon had performed the operation. There had been a moderate amount of discharge for three weeks. Sudden complete paralysis occurred twenty-four hours before operation. He thought osteomylitis of the temporal bone was present. The operation was advised because of the paralysis, no other symptom of mastoid disease being present. The thick non-cellular cortex explained the absence of the usual symptoms and why the disease had progressed inward involving the facial nerves in the petrous portion of the bone. The paralysis was entirely well in ten days.

THE SURGICAL ANATOMY OF THE TEMPORAL BONE. A LANTERN DEMONSTRATION.

By GEORGE E. SHAMBAUGH, Chicago.

Efforts to teach the surgical anatomy of the ear are restricted usually to the so-called operative courses on the cadaver. Experience has shown how inadequate such courses are for imparting that knowledge of the anatomical details of this region which the surgeon must have in order to operate about the ear without serious risk of injuring important anatomical structures.

Some years ago I worked out a series of anatomical preparations of the temporal bone, each preparation designed to bring out certain anatomical relations. The series included in all over fifty preparations. Some of them were copied from the most attractive illustrations in anatomical work on the ear. Most of the sections however, were planned originally to expose relations which had an important bearing for the surgeon in operating on the ear.

Efforts to expose in a single temporal bone one after another of the anatomical structures, destroying the one in order to expose another, fails to fix accurately in the mind any of these relations. It is only by the repeated handling of a series of preparations, each designed to bring out in the clearest possible way certain important relations, that the surgeon can hope to get such an accurate mental picture of the complicated anatomy of this region as will make him feel at home under all circumstances when operating on them.

In teaching from these preparations, I found that the quickest and surest way of fixing on the mind of the student the relations exposed in sections of the temporal bone, was to have the student spend a couple of hours making a careful drawing of the section and labeling the several structures

exposed with their correct anatomical terms. Two or three consecutive hours should be spent each day on this work. I have found that in six or eight weeks the student has usually been able to master the anatomy of the ear in such a manner that he will retain a mental picture of at least the more important relations. In the demonstration of slides I shall show a few of these sections.

Discussion.

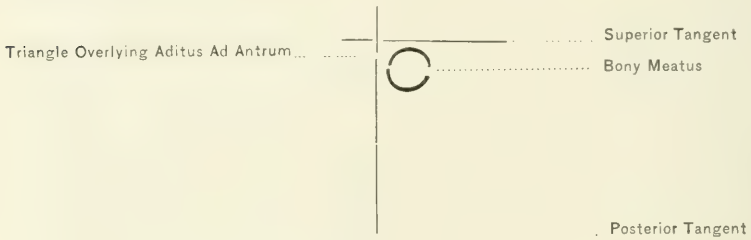
Dr. Harris Peyton Mosher, Boston, Mass., had been very much interested in what Dr. Shambaugh said regarding the teaching of anatomy. His own experience had been largely confined to the teaching of the anatomy of the nose and throat. It was very discouraging to give a student a preparation, have him look at it, throw it aside, and ask for more. For the last three or four years he had been using very much the same procedure for holding the student's attention as that employed by Dr. Shambaugh. For this purpose he had used an inclined box, with a glass front, by means of which about two-thirds of the labor of drawing was eliminated. He had found that the average student, with but little natural inclination toward drawing, would soon become discouraged if he had to sit for hours in order to get a reasonably good drawing. His enthusiasm would soon wane, and he would not persevere unless there was an examination, which was seldom the case in post-graduate work. By putting the specimen underneath the glass an outline drawing could be made quickly. This accomplished the details on drawing in free hand.

Dr. E. B. Dench, New York City, said, with reference to the deep cell beyond the digastric groove, he did not think it so uncommon and so often overlooked as one might imagine. It was separated from the other cells overlying it by a thin layer of bone, and was easily entered with the curet after removal of the cells at the mastoid tip. He had on one occasion accidentally opened the jugular bulb. In taking

away the floor of the meatus his chisel perforated the bulb. He controlled the hemorrhage by a small gauze pledget, isolated that part of the cavity with iodoform gauze and grafted the cavity ten days later.

He had always taught his students landmarks which would enable them always to go directly into the aditus and antrum.

If in any case a vertical line is drawn tangent to the posterior wall of the bony meatus, and another line is drawn horizontal and tangent to the superior wall of the bony meatus, the triangle thus formed and having for its apex the point of outer section of these tangents, and for its base that portion of the posterior superior meatal wall included between these tangent lines will be directly over the aditus ad antrum, thus:



Dr. William Sohier Bryant, New York City, thought the structures of the ear too small to lend themselves well to drawings such as Dr. Mosher described. He had found specimens better than drawings.

Dr. B. Alexander Randall, Philadelphia, emphasized the value of careful photography in the teaching and study of the structures under discussion. A good photograph gives with remarkable veracity details which, in a drawing, often have to be imagined. He laid particular stress upon the question of orientation,—a point often missed in teaching, and, when missed, leading not only to failure to teach but teaching wrongly. It was desirable and should be feasible, to make the vertical vertical, for example; but if this could

not be done it could be defined by lines. A picture out of line would mis-teach. He called attention, in this connection, to Politzer's picture, which is askew, giving a distinct outward deviation to the facial canal. This existed to very slight extent, except in anomalous conditions, such as that described by Dr. Berens. In ninety-nine out of a hundred cases this picture, if followed, would give a false idea. In placing these specimens on the screen it was important and feasible to present them in proper relation, or to so mark them as to prevent mistake.

He thought Dr. Shambaugh had not sufficiently emphasized the tubal cells, which are usually present, as also cells along the internal meatus, but in greatly varying degrees. Some textbooks say the latter do not exist, but he could show many specimens in which they may be seen, some forming the upper wall or other walls of the meatus, extending, in some cases, as far in as the sella turcica, some of them large pneumatic cells.

He considered it of great importance to teach developmental conditions, for example, that the membranous labyrinth exists before the bony labyrinth, that the facial nerve is present before the facial canal is developed, that the bony parts form around the soft parts, and that the soft parts are the constants and the bony parts the variants.

He had found it of great assistance in teaching students to see the drum-head to have them make a rude sketch of it.

Dr. Randall asked if those who used the supra-meatal *triangle* as a guide had not frequently found that it was a very good guide *to* the lateral sinus and hence the reason for reports of very anterior.

Dr. Wendell C. Phillips, New York City, always taught his students the necessity of retracting the tissues sufficiently far forward to reach the bony meatus. That would give the line to which Dr. Rae referred. He always taught them to go through the depression posterior to the spine of Henle. The spine was sometimes absent, though as a rule its site could be located.

Dr. Mosher added that he used to think the linea temporalis indicated this lower limit of his middle fossa. Experience has shown, however, that it is a true guide in only about a third of the cases. In two-thirds of the cases the base of the brain lies above this line.

Dr. James F. McKernon, the President, on behalf of the Society, thanked Dr. Shambaugh for the admirable demonstration.

Dr. John B. Rae, New York City, was of the opinion that the location of the Macewen triangle and of the Dench triangle presented certain difficulties to the student. The anatomical landmarks are frequently absent or so ill defined that the Macewen triangle is impossible of exact location. In tracing the Dench triangle the anterior flap must be retracted very far forwards in order to draw the tangents.

He has been in the habit for many years of teaching the following simple method. The anterior flap is retracted until the posterior, superior curve of the bony meatus is clearly defined. This curved segment becomes the base of an equilateral triangle which can (being equilateral) be completed without any further landmarks. This triangle conforms practically to both the Macewen and the Dench triangles and calls for only one landmark which can invariably be demonstrated.

A FURTHER REPORT OF CASES OF INFLAMMATORY AFFECTIONS OF THE LABYRINTH.

WENDELL C. PHILLIPS, M. D., New York, N. Y.

On Jan. 10th, 1913, in conjunction with Drs. Fowler, Kopetzky, and Sharp, I had the pleasure of reporting 20 cases of labyrinthine affections before the Sections in Otology of the New York Academy of Medicine.

Since that date a series of 6 cases have occurred in my various services, this series including one private case of Dr. J. J. Thomson, and one of Dr. J. C. Sharp. In the former series the diagnosis and treatment of the cases was given in full without comment or conclusions. The same rule is followed in the present series, it being my belief that there are certain points in the management of purulent labyrinthitis that can only be settled upon a basis of an extended series of cases.

I take this occasion to suggest to the members of this Society that they keep careful notes of every case, and report them in full, especially the autopsy reports, whenever autopsies can be secured.

Case I. S. Z., male, aged 52. Seen for the first time Sept. 9th, 1913. Nine years ago abscess in right ear, discharge began without pain, has continued since then, and has always been foul. Eight weeks ago attacks of vertigo in the mornings. Attacks increased in severity and four weeks ago nausea and vomiting with each attack accompanied by severe headache on right side. Compelled to remain in bed, unable to raise his head without vertigo, nausea, and vomiting. For past two weeks has felt better and has been up and about again. Complains now of slight unsteadiness in his gait, and headache. During the attack four weeks ago had fever and has lost considerable weight. He does not remember when he had hearing in his right ear. Mind clear. Recognizes and

can name objects presented to him. Pupils equal and react promptly to light. Knee jerks increased. No ataxia in arms or legs. No spontaneous pointing error. No adiadochokinesis. No paresis arms or legs. Functional examination. Right ear, total deafness. (noise apparatus). Spontaneous rotatory nystagmus to both sides, more marked to left. (sound side). Has tendency to fall to the right irrespective of the position of the head.

Rotation to right, Nys. 17 degrees—probably compensation nystagmus.

Rotation to left, Nys. 18 degrees.

Caloric Right Negative.

Caloric Left Rot. Nys. Right lasting 2 1-2 minutes (not prolonged).

Fistula test negative.

Operation. Radical mastoid operation and drainage of labyrinth (Sept. 12th, 1913).

Large cholesteatoma which had eroded tegmen of the attic aditus and antrum. Large exposure dura middle fossa which was covered with cholesteatoma matrix and granulations. Large irregular fistula in external semicircular canal, oval window does not contain stapes.

Postoperative. Pulse 70-80. Temp. 99.6-100.8. Headache persists, but patient appears better. Slight facial paralysis.

Sept. 30th. Headache persists. Pulse, Temp., eyes, normal. Appetite fair. Plastic with closure of posterior wound. Considerable gain in weight.

Oct. 7th. Very little headache. Temp., Pulse, Eye-grounds normal.

Oct. 10th. Patient left hospital. Condition good. Feels well. Radical cavity not completely epidermatized. For almost a month patient gained in weight, felt well, grew gradually stronger, and was considering his return to his work, but he did complain of more or less persistent headache, all other localizing signs being absent.

Nov. 6th. Suddenly intense headache, vertigo, nausea, followed by high fever and persistent vomiting. Readmitted to the hospital. Temp. 103, pulse, 86, head retracted, neck rigid,

cervical vertebrae tender, pupils sluggish, knee jerks increased, Kernig positive, delirium. Cerebrospinal fluid turbid, contains many leucocytes and cocci. The same night, chiefly for the reason that his headache had invariably been located in the temporal region, extending to the outer angle of the orbit, I decided to explore that region first. The same night evacuated a large temperosphenoidal abscess, pus green and very foul. After evacuation of abscess, pulse 120. Death in 24 hours from diffuse purulent menengitis. No autopsy could be obtained.

Case II. S. T., male, aged 24 years. Admitted to the Manhattan Eye, Ear & Throat Hospital Sept. 16th, 1913.

Chronic purulent otitis media Right for past 12 years. Discharge moderate in amount, and at intervals ceased entirely. General health good. Wasserman negative. One month ago cold and earache for two days, accompanied by an increase in the discharge. Felt suddenly faint and after a few moments vertigo and vomiting, which occurred at intervals for two days. Thereafter experienced vertigo whenever the eyes were rotated to the side. Vertigo also when stooping. Had an attack two days ago but at present is not dizzy.

Functional examination. Voice, Right, 8 feet.

Voice left, 2 feet.

Weber to right. Rinne both negative.

Spontaneous nystagmus toward both sides about equal.

Turning to Right, Nys. 16 degrees.

Turning to Left, Nys. 21 degrees.

Fistula test positive.

Operation. Radical mastoid operation. Large fistula in the anterior limb of external semicircular canal.

Following operation, partial facial paralysis. Hearing present, no increase in the spontaneous nystagmus.

Sept. 23d. Condition unchanged. No vertigo.

Sept. 30th. Plastic with suture of posterior wound.

Jan. 17th, 1914. Hearing present, whisper at few inches.

Rotation to right, Nys. 23 degrees.

Rotation to left, Nys. 29 degrees.

Caloric positive. Fistula test negative. Facial paralysis subsiding.

Case III. E. C., female, aged 10 years, patient of Dr. Sharp. Chronic purulent otitis media, right, since infancy. Discharge rather profuse and foul. No tenderness of mastoid, no edema. Fundus full of granulations.

Functional tests: Right ear, hearing totally destroyed. (noise apparatus).

Caloric positive. Fistula test negative.

April 16th, 1914. Radical mastoid operation was performed by Dr. Sharp in the Manhattan Eye, Ear & Throat Hospital. No fistula in the labyrinthine capsule. Cavity not yet epidermatized. Hearing entirely destroyed.

Case IV. E. S., male, aged —, admitted to the Manhattan Eye, Ear & Throat Hospital Jan. 28th, 1914. Dr. Kopetzky had charge of the case.

Family history negative. Previous history: Has always been well and strong. At eight years of age was operated upon for a "swelling" behind the right ear. This ear was reoperated upon when he was 16 years old. Since that time the ear has discharged constantly but not profusely. For the past few days slight headache. Patient presents a partly epidermatized radical cavity. Inner tympanic wall contains few granulations. Some discharge. No tenderness.

Functional tests: Left ear hearing normal.

Right ear total deafness, (noise apparatus).

Caloric positive. Weber to left. Schwabach right shortened. Rinne right negative ad infinity.

Spontaneous nystagmus to both sides, more marked to left. No Romberg or ataxia.

Rotation to right Nys. 20 degrees. Compensation. Rotation to right Nys. 18 degrees.

Caloric right negative. Weber to left. Fistula test negative.

Secondary radical operation by Dr. Kopetzky. In the region of the tube granulations and polypi. The inner tympanic wall consisted of dense newly formed bone which had

obliterated foramen ovale, the foramen rotundum and the entire labyrinth. Small exposure of sinus. Plastic and suture. The radical cavity is now completely epidermatized.

Case V. A Case of Labyrinthine Fistula. I am permitted to report this case by the courtesy of Dr. J. J. Thomson.

M. C., female, aged 18 years, was seen for the first time Jan. 16th, 1914, and gave the following history. Has always been a healthy girl, and with the exception of measles, chickenpox, and whooping-cough, has not suffered from any of the usual diseases of childhood. She stated positively that until six weeks previously, she had never had any trouble with her ears. Her father and mother, in spite of repeated questioning also were positive that she had never had discharge, or even an earache. No family or personal history of tuberculosis could be obtained.

Six weeks ago she had a severe cold followed by acute otitis media in the left ear. The drum ruptured spontaneously, and the ear has discharged profusely since. She has not had any marked pain since the rupture of the drum, but the mastoid has been tender at times for the past four weeks. She has had tinnitus and defective hearing in the left ear since the beginning of the otitis. The day before her visit to me, she noticed that she was a little dizzy when she turned her head rapidly, and the morning of Jan. 16 was more so, but was able to be about, and only felt dizzy when she turned her head. She had not vomited or felt nauseated.

Exam. The drum could not be seen, on account of narrowing of the canal at the fundus. There was what appeared to be a granulation at the bottom of the canal. There was a moderate amount of discharge not very malodorous, culture from which grew staphlococus arreus only. The mastoid was very tender, but not swollen. Temperature was 99.4, and pulse 88. There was hearing in the left ear, but only a loud voice could be heard, and she lateralized the Weber to the left ear. The caloric test was negative, but I think this was due to our inability to get the water far enough in the canal. There was a very marked positive fistula test. Compression

would cause a rotatory nystagmus toward the diseased ear which would last for several minutes. There was no spontaneous nystagmus.

The following day the same tests were made, and gave the same result. All the symptoms were the same. It was decided to wait a few days before doing any operative procedure, on account of the recent onset of the dizziness. Jan. 18th she vomited a few times and felt nauseated a good deal, and said for the first time that she felt dizzy while lying quietly in bed. There was also a slight spontaneous nystagmus toward the good ear.

Operation. The mastoid cortex was removed with the rongeur without the use of a chisel to avoid jarring. On its removal, a whitish membrane presented in the cavity which resembled somewhat the lateral sinus, but while we were observing it, pus began to exude from under it under pressure, and it proved to be a membrane enclosing about two drachms of pus and granulations. Culture from pus did not grow any bacteria. This was a large perisinus abscess. A radical operation was done. The incus was not seen, but the bleeding was so profuse that I could not tell for sure that it was not removed without being observed. A portion of the malleus was recovered, and the stapes was seen in place. There was a large fistula in the horizontal semicircular canal, occupying the position of the maximum of its convexity. This fistula was covered by a pad of granulations.

The evening of the operation there was no change in the nystagmus. It was still directed to the sound ear and was apparent on the slightest rotation to the right, but not when the patient looked directly ahead . During the next twenty-four hours the patient was comfortable, there was no dizziness or nausea, or rise in temperature.

Jan. 21st. Patient is feeling well, has good appetite, has not been dizzy and temperature is normal. The spontaneous nystagmus is less marked than before operation but is still present, and is directed toward the sound ear. In the Weber test the sound of the fork is heard in the operated ear.

Jan. 23d. First dressing. Wound is clean, temperature normal, no dizziness, and the spontaneous nystagmus has disappeared. Weber lateralized to the operated ear. Patient is allowed to sit up.

Jan. 31st. Plastic operation performed. Posterior wound closed. Stitches became infected lower part of posterior wound. Wound opened.

Feb. 16th. Wound doing well. Middle ear epidermatermized.

Feb. 25th. Wound entirely healed and patient discharged. Hearing distance for the whisper in the operated ear is 20 feet.

Case VI. Report of a Case of Labyrinthitis Suppurativa.

This case came to Dr. Phillips' Clinic at the Manhattan Eye and Ear Hospital April 5th, 1913. He gave a history of dizziness for about two weeks. He had chronic suppuration for a great many years in both ears. Both ears discharging slightly at examination. Patient did not complain of anything but dizziness and deafness. Tests:—Hearing in both ears tested with noise apparatus. Very little hearing in right ear. Rotation to right, Nys. to left for 14 seconds. Rotation to left, Nys. to right for 9 seconds. Caloric test negative in right ear, not used in left. Patient sent to hospital. A few days later he complained of severe headache, and had a temperature of 100. Neither mastoids were tender, and the discharge was less in each ear. Tongue coated and remained so in spite of free catharsis. The severe headache continued for a week, the patient was rational, memory good, eyegrounds clear, temperature from 100 to 102. Pulse gradually became slower until it was 60, but at times would go to 88. No retraction of head, dizziness disappeared, headache improved after ten days, but returned. No leucocytosis, count negative, headache variable, but usually some there, localized to the left side. No vomiting. Patient gave a history of syphillis, and has old cutaneous lesions. Wasserman was negative.

Examination on April 18th showed very little change. Patient rational and clear. Memory good. No pain or per-

cussion of head. No mastoid tenderness. No dizziness. Nystagmus spontaneous to the left. Hearing for watch apparently present in right ear. Hearing in left. No restlessness. Headache still present. Tongue coated. Temperature 102. Had a chill in the morning. Exploration of the cerebellum the following day. Decided on Syphillitic treatment. Also ordered spinal puncture. Examination of fluid showed it to be loaded with streptococcus mucosus. Chemical tests showed excess lactic acid, absence copper reducing agent. Patient almost unconscious. Facial paralysis present. Condition very bad. Operation Drainage of Cisterna magna. Cerebellum directly under dura. Only small amount of fluid escaped. Patient died same night.

Autopsy. Mastoid filled with pus—purulent labyrinthitis—meningitis—no abscess.

Autopsy of Head Only Permitted. Removal of the cranial vault shows that the dura is very adherent to the bone throughout its extent.

The meninges tissues are thickened—evidence of a chronic inflammatory process being presented.

The opened meninges revealed a large collection of thin fluid in the meshes of the Pia. The largest collection of this fluid is found in the Posterior Cranial Fossa. The membranes at the base of the brain generally are more acutely inflamed than elsewhere.

Removing the brain, we find a rather distinct collection of whitish pus—in contrast with the other collections of fluid found—localized at the Internal Auditory Meatus, filling this structure and bathing the facial and the auditory nerves.

The removal of the adherent meninges from the bone on the internal side of the petrossa, discloses darkly discolored bone, involving the tegmen tympani, and tegmen cillulae mastoidea.

Opening the tegmen in these regions, finds the tympanic cavity filled with purulent contents, semi-fluid in nature. The malleus is recovered and found to present no evidence of necrosis. The incus is not found. The removal of the tegmen cellulae mastoidae finds the mastoid process filled with dark,

discolored, thick, but semi-fluid mass—some intracellular walls are present, but the probe easily breaks these down-evidence of an acute infection of the mastoid process.

Opening inward into Cochlea from the internal auditory meatus, we find this structure likewise filled with similar purulent matter, the only anatomical structure intact being the promontory. The Superior semi-circular canal is likewise discolored. The lateral sinus on the right side contains a red thrombus—probably a post-mortem condition.

The Brain. Pial vessels injected. Both lateral ventricles much distended with a water-clear fluid. Incision into sections of both the cerebrum and the cerebellum fails to reveal any abscesses.

Comments on Autopsy Findings.

The Autopsy finding warrant the following conclusions:—

1. That the patient suffered from an old chronic meningeal inflammation. His admission of having had syphillis, would explain this finding, in spite of the negative report of the Wasserman.

2. There was an acute exacerbation, or an acute infection of the middle ear and the labyrinth cavities. The intact malleus, with its ligamentus attachments, speaks against a chronic middle ear infection. The bacterial finding also speaks against a chronic otitis media. The acute infections involved the middle ear, including the mastoid process, and the labyrinth, finally breaking endocranally along the route of the nerves, facial and auditory, to begin the infection of the meninges at the internal auditory meatus.

3. The infection resulted in a general involvement of the basilar meninges, and the accumulation of purulent fluid in the posterior cranial fossa—a posterior basilar meningitis resulting. The distension of the lateral ventricles by water-clear fluid was a secondary manifestation.

4. The facial paralysis noted as presenting itself on April 19th, just prior to operative intervention of the meninges, is accounted for by the route travelled by the infection from the internal ear to the meninges.

5. The operation for the drainage of the Cisterna Magna was not successful, in so far that there was present at autopsy a very large collection of pus in the posterior cranial fossa, which was not drained off by the procedure, although this opening did not sufficiently divide the foramen magnus from the internal side.

(Note). There is no way of telling whether this accumulation of fluid in the posterior cranial fossa was an artifact of post-mortem incident, or not. Certainly at the time of operation there was no such collection of fluid present as the cerebellum presented itself immediately upon incision of the dura.

Discussion.

Dr. E. B. Dench, New York, confirmed what Dr. Phillips had said regarding the necessity for very careful examination in these cases, emphasizing the fact that such examinations require more time than can ordinarily be devoted to hospital cases. However, by devoting this time, and by giving careful functional tests in all cases with a history of suppuration, a great deal of valuable data would be revealed. The last case mentioned by Dr. Phillips suggested one in which he had operated early in the fall. There was a dead labyrinth, with a history of suppuration late in the summer. There was also a history of syphilis. It was a case of perilabyrinthitis. After clearing out everything in sight the labyrinth regained its function and the hearing returned. He was not satisfied with making this examination alone, but had an assistant to check up every point. There was absolutely no hearing. At the present time the patient could hear a whisper twenty-three feet.

The Germans advocated taking out the labyrinth in all cases in which it is dead; he did not think this always necessary. He always removed the softened margins around a fistula, and then waited. In a good many cases extirpation of the labyrinth might be followed by meningitis and death. According to the latest reports of Alexander, some tem-

perature, say 101 degrees, and headache, in addition to dead labyrinth, were considered necessary indications for extirpation of the labyrinth.

In one case, in which erosion extended to the labyrinth, he had been tempted to take out the labyrinth, but by enlarging the opening in the semicircular canal the man made a perfect recovery, having had no headache since the operation, which was done two or three months ago. The pendulum seemed to be swinging backward, and fewer labyrinth operations were being done than was the case a few years ago. He had yet to do his first radical operation in non- suppurative labyrinthitis.

Dr. George E. Shambaugh, Chicago, stated that there are a great many cases of labyrinth disturbance not associated with suppuration of the middle ear. Very often these cases consult the internist because of the attacks of nausea which they attribute to some disease of the stomach. In a good many of these cases, the labyrinth origin of the disturbance is clearly indicated by the associated tinnitus and deafness, but in not a few of these cases symptoms pointing to an involvement of the cochlear branch of the eighth nerve are very slight or are absent entirely. Cases of this sort where the attack of vertigo is not associated with tinnitus or deafness are not infrequently the result of a toxic neuritis which involves only the vestibular nerve. In other cases the neuritis may involve the cochlear part of the eighth nerve alone or in connection with the vestibular portion. The cause of toxic neuritis of the eighth nerve is the same as the cause of neuritis involving other nerves. Dr. Shambaugh called attention to the great frequency with which neuritis is caused by infection, acute as well as chronic, involving the faucial tonsils, and referred to several cases where neuritis of the eighth nerve could be traced clearly to infection in the faucial tonsils.

Dr. B. Alexander Randall, Philadelphia, took a more conservative view with reference to these cases, a view which

was growing. He had seen in the Menière type of cases very notable improvement which he had ascribed to medication. He had seen pontine symptoms clear up very nicely without operation upon the labyrinth. He cited the case of an elderly woman, with a grippal history. A general surgeon and one of the best neurologists had gone carefully over her, and the question arose whether a labyrinthine or an intracranial operation should be done. He spoke in favor of the latter. The symptoms at the pontine angle had been slight, but the trouble was sufficient to cause the two nerves in the auditory canal to be so tightly bound together that it took the surgeon half an hour or more to separate them. This was a case of peri-neuritis. Cases with these symptoms were quite numerous, and he thought a little conservatism not misplaced. Alterative treatment would bring many of them around all right without operation.

In connection with the matter of drainage in the cases of meningitis, he called attention to the value of making the opening through the squamos portion of the temporal bone. Drainage by the cisterna magna operation had never appealed to him, as had temporal drainage, which had proved so effective that within five minutes after beginning a hypodermoolysis the temporal dressing would be soaked.

Dr. Phillips, in closing the discussion, said he had eight assistant surgeons and four clinical assistants on his staff, and previous to two or three years ago he recalled how little time was spent in the testing room. Now, however, hardly a day passed that the room was not occupied in testing suspicious cases of labyrinthine affections. It was only in this way that valuable material could be accumulated. If one could ever reach the point of doing anything for the relief of non-purulent labyrinthitis it would be well worth while. He cited the cases of two sisters, in the late thirties, each of whom had had tinnitus, slight deafness and severe attacks of vertigo, for many years. They had been under the care of a confrere in Washington, but nothing seemed to give the slightest relief. He wondered if the condition was not caused

by a bony growth in the locality. One of the women had promised that he should have her temporal bone in case of her death.

He had been very much interested in the discussion with reference to the outcome of operation. So far as his observations had gone the ultimate results in acute purulent cases were rather better when the labyrinth was not opened. Those who advocated conservatism were right in doing so; at the same time, there was only a short period in any case during which it was worth while operating at all. Upon the advent of purulent meningitis it was absolutely useless to operate.

AURAL COMPLICATIONS IN TYPHOID FEVER.

By EDGAR M. HOLMES, M. D., Boston, Mass.

Twenty years ago I studied the ears in 234 patients ill with typhoid fever at the Boston City Hospital, and 17 years ago I presented a paper at the annual meeting of the Massachusetts Medical Society, giving the results of these investigations. Although I have never been able to study the ears in so large a number of typhoid patients as a group since that time, having served as an aural surgeon to a large general hospital, I have been called upon to examine many patients, who had developed aural disease during an attack of typhoid, or who suffered with a pre-existing pathological condition of the ear which was aggravated by the typhoidal condition. There have also been many cases presenting typhoid symptoms and signs, which proved to be due to complicated aural sepsis and not to typhoid bacillus.

In considering the subject, we have to deal with the aural complications which may develop during the course of the typhoid infection, those which have existed before the onset of the typhoid condition and those cases of aural disease which through extension into the lateral sinus or brain may simulate in many ways the symptoms of the typhoid state when there is no typhoid infection present. This latter class is a very important one, both because of its extreme severity and high mortality, and because early recognition and skillful surgical interference can often save the life and enable a restoration to comparative health of those almost surely doomed to death, if left to nature or to unaided medical treatment. It is quite generally known, that during an attack of typhoid fever, there is liable to develop inflammatory conditions within the ear, that there is often a state of deafness more marked than the aural signs or the mental depression would account for, and that these cases are liable

to be refractive due to the lowered state of the patient's vitality. The time allowed for the presentation of this subject, is not sufficient to consider the various aural complications at all thoroughly, therefore, the main purpose of this paper is to again impress upon the physician who is responsible for the care of a typhoid patient, the great importance of not forgetting the liability of aural complications in this disease, and of their seriously affecting the progress of the case. It is fully as important to remember, that a grave aural complication may be the one and only causative agent in producing a symptom complex which may so closely resemble a true typhoid infection as to, for a time, deceive the skillful clinician unless he pay strict attention to the ear and its associated organs, the lateral sinus and the jugular vein. A patient may have had a typhoid infection and have presented all of the signs of that disease and may, during the height or at the later convalescence of this disease, develop a purulent lateral sinus and jugular thrombosis with all of the dangers of these complications. Not only is lateral sinus thrombosis frequently mistaken for typhoid fever, but it is a comparatively frequent complication of this disease and probably no complication has been so frequently overlooked. There is good reason why this condition can so frequently escape the attention of the clinician. One is that the great majority of these patients are extremely ill and they are very much depressed mentally as well as physically. They are often too ill at first introduction to the physician, to be able to give a clear history of the onset of the disease, or of their past ailments and frequently, the friends know comparatively little about the previous physical condition of the patient. This is especially true in regard to a previous aural disease, which may have been comparatively slight and of short duration. There are many who have had a low type of middle ear for years, who scarcely pay attention to this aural condition and there are some who even are not aware that such trouble exists. Again there are a few who may have an infection within the ear extending into the mastoid and to the lateral sinus, without producing the usual symp-

toms of middle ear disease and without leaving any signs in the drum membrane of any disease. One of the cases reported at this time belongs to this class, and the condition was diagnosed by the thickened feel of the jugular and the septic temperature chart. Many of these cases could go to autopsy and unless especial attention were paid to the lateral sinus and jugular the true cause of death might remain unknown. This is especially true in cases where permission to open the head cannot be obtained, and the lateral sinus cannot be examined. One of the most important considerations in this condition is the diagnosis, for if a positive diagnosis can be made there is no serious question as regards treatment. An operation to tie the jugular vein and open the lateral sinus is imperative and immediately necessary, and today every aurist is familiar with the technic and should possess the skill to thoroughly cope with this condition. It is unfortunately not always possible to make an absolutely positive diagnosis in this condition. It is especially hard in many of the cases which complicate a true typhoid condition, or which develop shortly after the extension of a typhoid infection, for there are so many conditions complicating typhoid as well as the proneness of this infection to the so-called relapses which may give increased temperature and other signs of sepsis. If there are signs of middle ear infection or mastoid extension, and we have had symptoms of pain and tenderness about the ear, or if there is tenderness of the carotid sheath associated with a septic temperature with or without chills, a diagnosis can reasonably be made, but when we have chills and accompaning temperature without pain, without a history or signs of a middle ear inflammation and when a blood count shows only 11,000 whites and when there is no jugular tenderness and there are no mental or opthalmic signs suggestive of septic thrombosis, we have to consider and eliminate, if possible, the other frequent complications of typhoid before giving too much weight to the probability of septic lateral sinus thrombosis. In fact, it is almost as serious a blunder to be misled and operate upon a healthy mastoid and lateral

sinus in a patient suffering from some other complication of typhoid fever, as it is to overlook and not operate upon a septic sinus thrombosis. There is one very positive and valuable diagnostic sign, which is often present when sinus infection has extended into the jugular. There is phlebitis which shows a distinct change to careful palpation. In order to obtain the best results in palpation of the jugular, it is necessary to have the neck muscles, especially the sterno cleido muscle, as lax as possible and the sheath of the large vessels must be palpated with delicacy and care. It is always advisable to carefully compare the jugular veins of the two sides, and yet to not forget that in health there is sometimes some variation. If the patient is not too apathetic this careful palpation will often demonstrate a hypersensativeness of the jugular sheath. This sign if present, is a very valuable one but its absence does not in the least eliminate the possibilities of a septic sinus or perhaps even of a jugular phlebitis. We must also if possible, be quite sure that the tenderness is in the jugular sheath and not in the cervical glands, for the superior deep cervical glands are often infected and sensitive when there is a simple ear, Eustachian tube, or epipharyngeal infection.

Much has been written concerning leucocytosis, but in these cases it has been of little help. In one of our severe cases we had 11,000 and in another about 18,000. A marked polymorphia leucocytosis when present would add to the positiveness of our diagnosis in cases where other signs are vague or absent. It is of considerable importance not to place too much weight upon the blood findings, either positive or negative, though they may be of help when considered with other signs. Frequently the patient is not seen until the condition is well advanced. This is especially true of hospital cases, as many cases are treated at home or have no treatment until they are becoming very severe, when they are referred to or seek aid at a general hospital. We may have meningeal extension with the associated headache and, perhaps, eye symptoms. There are also frequently foci of metastatic infection. These may occur in the lung, spleen,

heart, liver, joints or in the brain and the symptoms and signs will be such as occur when any of these various organs become affected. These may be more prominent than those arising from the primary disease. It is the late complicated case which can be so liable to be diagnosed as typhoid fever. Typhoid patients are also very liable to develop aural disease, or have exacerbations of pre-existing aural disease during the course of the typhoid. These cases having a positive widal and other positive symptoms and signs of typhoid often complicate and add to our difficulties in making a positive diagnosis of any co-existent aural complications. It is not rare to find not only a septic thrombosis, but frequently so long a time has passed since the onset of the sinus complication that the thrombus has been broken down and the sinus has become filled with a necrotic purulent mass which only a small distal clot within the jugular may prevent from entering the circulation. The liability of this condition, makes it important to first tie the jugular beyond the thrombus if possible, before attacking the sinus within the cranium. The possibility of this grave condition complicating a typhoid infection, makes it extremely important to carefully watch the ears during an attack of this disease and especial attention should be given this subject whenever any unaccountable symptoms of sepsis arise. We, as otologists, are rarely called to these cases at first, nor are they usually admitted to our aural clinics or special hospitals. The family physician and the general medical hospitals first meet the great majority of them. It is our duty to not only impress the importance of this subject upon the general practitioner, but to become as proficient as we possibly can in caring for them when called in consultation, either in private or in general hospitals. With all the knowledge and care at our present command, there will be some cases so obscure and complicated, that we shall fail in our diagnosis, and in this failure must blunder in operating or in waiting. It is a grave responsibility, and any work which can add to our knowledge, will be of the utmost importance.

From Oct. 1st, 1913, to Feb. 1st, 1914, I was able to diagnose and operate upon five cases of septic sinus thrombosis at the Boston City Hospital, in patients who were admitted as probably typhoid. Although these cases showed many symptoms of typhoid fever, they were not typical and the physician in charge called me in consultation for an aural examination. The widal test was negative in four cases, and the three-hour temperature chart carried for several days, suggested septic absorption in all of them. There were signs of beginning choked discs in two of these cases.

R. C., a man 34 years of age, of powerful physique, was admitted to the second medical service at the Boston City Hospital, Oct. 19, 1913. He had contracted chancre 14 years previously and had had an abscess in the right ear in July, 1913. Since July, frequent attacks of headache and three weeks before admittance a severe chill. Severe headache and fever, off and on for three weeks, some abdominal pains and soreness and slight hacking cough for over a week. There had been dyspnoea for a week and vomiting for three days. Physical examination showed eyes, throat, heart, and lungs to be normal. The bowels were lax and tympanitic. The reflexes were normal and there were no Babinski or Oppenheim. The blood count showed 20,000 whites and a culture of staphylococcus pyogenes was obtained. On Oct. 22 in consultation the right ear showed much foul pus. The membrane and part of the handle of the malleus were gone. There was tenderness in the digastric fossa and the jugular was distinctly thickened. There was some retinal stasis. The pupils were normal. The left shoulder and right knee were tender and somewhat swollen. Immediate operation was performed. The jugular was filled with thrombus and it was necessary to tie below the facial vein. The lateral sinus was filled with suppurating clot to nearly the torclea. Following the operation, the local condition progressed favorably, but there were several septic joints and two attacks of facial erysipelas. He was not able to be discharged from the hospital until Nov. 30. He has been perfectly well since.

A. H., a girl 13 years of age, was admitted to third medical service. Nov. 28. 1913. She had suffered with rheumatism when five years old. Was fairly well till 14 days before admission, when she had a severe chill. There were pains all over body, especially in back. At first there was ear ache. Had pain about heart and loss of appetite. She was apathetic. The tongue was dry and heavily coated. The teeth were covered with sordes. There was a systolic murmur and the apex was in nipple line. The other organs were normal. The blood culture was negative and the white blood count was 22,000. An examination showed the ear to be apparently normal. The mastoid was slightly tender. The jugular was distinctly thickened. I operated Nov. 29. The jugular was filled with thrombus and a low tie was necessary. The mastoid was filled with granulation tissue. The sinus was thrombosed to near torclea. After the operation the symptoms were much relieved for five days, when symptoms of general meningitis developed. Decompression relieved for 48 hours when she grew suddenly worse and died.

N. E., an Italian, 36 years old, was admitted to first medical service, Dec. 5, 1913. As far as known, he had never been ill until two weeks previously. He began with headache frontal and occipital. There was at first, slight earache. He was much prostrated. The abdomen was tympanitic. The white count was 41,000. There was rigidity of neck, the right ear was discharging pus, the anterior inferior quadrant of membrane was gone. The jugular was normal to palpation. There was a slight Kernigs, increased knee jerks and Babinski both sides. The white count in spinal fluid was 300 per MM. A mastoid operation showed the whole bone filled with granulation and pus. The lateral sinus was exposed and found to be thrombosed. The jugular was tied and the sinus and bulb cleaned out. There was hypertension of the dura. There was four days relief following operation, when the symptoms of meningitis returned and the patient died seven days after the operation.

B. B., a printer by trade, admitted to hospital Dec. 4, 1913. He had been ailing for several weeks and treated for typhoid. There had been severe headache and stiff neck. The day before admission, there had been three chills. Examination showed normal reflexes, negative widal, blood count 14,000 and a general septic appearance. There was tenderness over left mastoid tip and fluctuation below tip. The jugular was infiltrated. The membrane was red and bulging. There was sagging of posterior wall. An operation was performed and the mastoid found filled with pus and granulation tissue. The sinus was thrombosed and the dura much inflamed and bathed in pus. The case seemed helpless and the chances were that there was a general purulent meningitis. The patient died six days later.

Miss V. P., admitted Nov. 14, 1913, complaining of blurred vision, weakness, headache, loss of appetite, nausea and vomiting, chilly sensation, prostration. When a baby had abscess in right ear. When fifteen years of age the same ear discharged for a while. The case was diagnosed as typhoid and sent to the hospital. Physical examination showed considerable emaciation. The heart and lungs were normal. The bowels were tympanitic. The white count was 20,000. The patient continued with many typhoidal symptoms, but there was no widal and there were no typhoid bacilli in the stools. I examined patient Dec. 10, and found a chronic suppurating right ear. There was a spontaneous nystagmus to the left. Pressure on tragus increased this and produced marked vertigo. The jugular was distinctly thickened. The following day the jugular was tied, the mastoid was cleared and the outer wall of the horizontal semi-circular canal was found to be gone. The sinus was filled with thrombus half way to torclea. The canal of the facial nerve was very necrotic and nerve was exposed 1/3 of an inch. Following the operation, there was relief from symptoms. There developed partial facial paralysis which slowly cleared up. The patient was discharged. Relieved on Jan. 2, 1914.

It is evident that all of these cases were advanced lateral sinus thrombosis, and had the medical attendants correctly diagnosed them, or had they had the help of an otologist, the true condition would have been found and thorough operation would have saved more of them and, perhaps, could have saved them all.

Discussion.

Dr. Harris Peyton Mosher, Boston, Mass., thought the title of the paper should read, "thrombosis of the lateral sinus simulating typhoid fever."

Dr. B. Alexander Randall, Philadelphia, said in reporting, some years ago, a case of phlebitis of the lateral sinus in typhoid fever Dr. Keen had told him that this was the only case of such phlebitis of the lateral sinus with which he was acquainted. True typhoid phlebitis must be rather rare.

Dr, Ewing W. Day, Pittsburg, Pa., said he and Dr. Jackson, several years ago, when Pittsburg held the typhoid fever banner, had studied over eight hundred cases, five per cent. of which had ear complications. They could mark out pretty clearly when the complications would come on, which was generally at the beginning of the fourth week. In cases of typhoid running a high temperature, and with middle ear complications, if the foot of the bed was elevated mastoiditis would invariably develop. They found no case in the eight hundred in which the complications involved the lateral sinus. The study of these cases led to the conclusion that the matter of aural complications was merely a question of lowered vitality, which allowed the microorganisms to accomplish their work.

Dr. Holmes, in closing the discussion, said three or four per cent. of the cases showed toxic neuritis, with lowered hearing, which was not necessarily proportionate with the mental

dullness, and which might last several weeks after the mental condition had subsided. Referring to the question of laternal sinus thrombosis, he said only one of the cases was true typhoid, the others having been admitted as typhoid but not being so. Five or six per cent. of the cases of typhoid during the year had had lateral sinus thrombosis. He could not understand why there should be more cases of phlebitis of the lateral sinus in Boston and New York than in the West.

CONGENITAL BILATERAL MICROTIA WITH TOTAL OSSEOUS ATRESIA OF THE EXTERNAL AUDITORY CANALS, OPERATION AND REPORT OF CASES.

By JOHN RANDOLPH PAGE, M. D., New York, N. Y.

The attitude of writers on this subject in the past has been, with few exceptions, so universally against operation that without doubt a considerable number of skillful surgeons have been influenced by it to refrain from operation on cases that could have been materially helped. And although such deformities occur only in one[1] out of two thousand cases, the unilateral being six times as frequent as the bilateral,* they are sufficiently numerous to warrant some attention being directed to them, particularly when it is found, as I believe investigation will show, that it is the rare exception where improvement in hearing will not be gained by the proper surgical procedure. The possibility of gaining for the child the early development of speech and expression and all that goes with improvement in hearing is well worth striving for.

Nevertheless a review of the literature shows an almost unanimous opinion against operation on a young child for microtia with total atresia, three reasons being repeatedly offered to support this opinion:

First, that where the external ear is deformed and the meatus congenitally occluded there invariably exists such extensive defect either in the conducting mechanism, or in the perceptive apparatus, or both, that operation is unwarranted.

Second, that the operation should not be performed unless *tuning fork and bone-conduction tests* are found to indicate a functionating labyrinth.

Third, that it is practically impossible to prevent closure of an artificial canal after it has been made.

*Statistics quoted by Imhoffer[1], Bezold being his authority.

A strong argument against the first reason is found in the development of the temporal bone itself. For while the deformity of the auricle, canal and cartilaginous portion of the eustachian tube, is explained by faulty closure of the first branchial cleft with arrest of the unrolling process of the auricle during its development,[2] etc., there is found in the development of the temporal bone that the bony meatus is not at all associated with the development of the *essential organ of hearing*, [3] and that it and the labyrinth are developed from two separate and distinct origins. Furthermore, the greater part of the tympanum itself, including the osseous portion of the eustachian tube, except a small part of the anterior tympanic wall and roof, develops with the essential organ of hearing in the petromastoid portion, and is not associated with development of the meatus and auricle in any way.[3] In Politzer, (page 30), we find the following stated: "According to Gradenigo, the inner portion of the foot-plate (of the stapes) is developed from the labyrinthine capsule, while the external part is derived from a ring-shaped deposit, which is situated laterally to the labyrinthine capsule." This explains the fact that the stapes is rarely found missing in these while the other ossicles may be.

Even in cases where there is total absence of the eustachian tube accompanied by absence of an external meatus and the tympanic cavity, a normal labyrinth[5] will usually be found, and in all probability a stapes.

In support of this Alexander[6] expresses the conclusion, reached after many autopsies on cases with congenital deformity of the auditory tract, that in the vast majority of such cases the deformity is limited to either the conducting apparatus alone, or to the perceiving apparatus alone. Practically never is the defect present in both at the same time. He makes exception only in cases of non-viable embryos in which deformities of all three divisions of the ear have been found, and says that in such cases the deformity is always associated with other grave abnormalities elsewhere in the head, or heart, or vascular system. For further evidence he found, in the examination of a large num-

ber of congenital deafmutes no cases of atresia of the canal and none with deformed auricles, and in the twenty-five cases of congenital atresia functionally tested by him there existed not a single case of total deafness.

His statement that in all cases of congenital atresia an antrum exists, and that the mastoid is unusually large is interesting from a surgical standpoint, though this does not agree with the observation accredited to Michel, by Politzer,[7] that in complete absence of the external and middle ear one generally meets with non-development of the mastoid process or even with its complete absence. Nor do his other observations agree entirely with those of the numerous gentlemen quoted in Politzer's work. However, a consideration of the embryology of the ear inclines one to favor Alexander's conclusions rather than those expressed in Politzer.

It can definitely be stated then that cases of defective development of the internal ear are rarely, if ever, found to be associated with atresia of the canals in otherwise well developed infants.

An interesting observation accredited to Alexander by Castex and Berruyer[4] is that malformation of the labyrinth always affects the facial nerve and auditory at the same time, and that one can conclude from this that all malformations of the external ear coexistant with facial paralysis indicate alteration in the labyrinth.

The following is a brief account of the operation and findings in eight cases, four of which are from the Manhattan Eye, Ear and Throat Hospital, the others are taken from the literature. Unfortunately we are unable to ascertain the findings and results of anything like the total number of cases that have been operated on, for, although undoubtedly a large number of such cases have been tampered with surgically, they have not been reported.

Alexander's case[6] in a boy eleven years of age with a normal left ear, and congential microtia with total bony atresia in the right is interesting, despite the fact that the operation was performed not primarily for the relief of deafness, but for acute suppurative mastoiditis in the deformed ear follow-

ing Scarlet Fever. The findings at the operation (February 17, 1907) were as follows: Complete absence of bony canal, pus under pressure throughout the mastoid cells, and a remarkably small antrum The ossicles were present, the drum-membrane absent, and in its place there was a bony plate. The anterior wall of the wound cavity was in contact with the maxillary joint. The description of the operation did not state whether the ossicles were removed or not. He completed his operation by tucking in square shaped skin flaps from the posterior part of the wound. The meatus thus formed he says was packed, and eight months later it remained open to a diameter of 4 millimeters. Conversation could then be heard through a tube at 4 or 5 meters, while before operation it had been heard at 1 meter.

Blumenthal,[8] in reporting two cases operated on by himself, attributes the improvement of hearing in this case of Alexander's to the relief afforded the suppurating mastoid, and not to the formation of a meatus and canal to the tympanum. The results in his cases were evidently unsatisfactory 'though of this he does not speak definitely, nor does he give the age, functional tests, or findings at operation in either of his cases. As an argument against operation, however, he mentions a case of Brühl's, a school boy with bilateral microtia and atresia who could hear a whisper at one-half meter and conversation at 2 meters, and was able to attend school without having to be placed in a class for those with defective hearing.

The possibility of the existence of a fair degree of hearing is an argument occasionally offered against surgical interference in these cases, but in the light of late experience and improvement in the radical operation it seems that this degree of hearing is an indication for rather than against operation, for after all even in the most favorable cases where operation has not been performed the hearing is still decidedly limited, and very marked improvement might be reasonably expected in such cases after the performance of the proper operation.

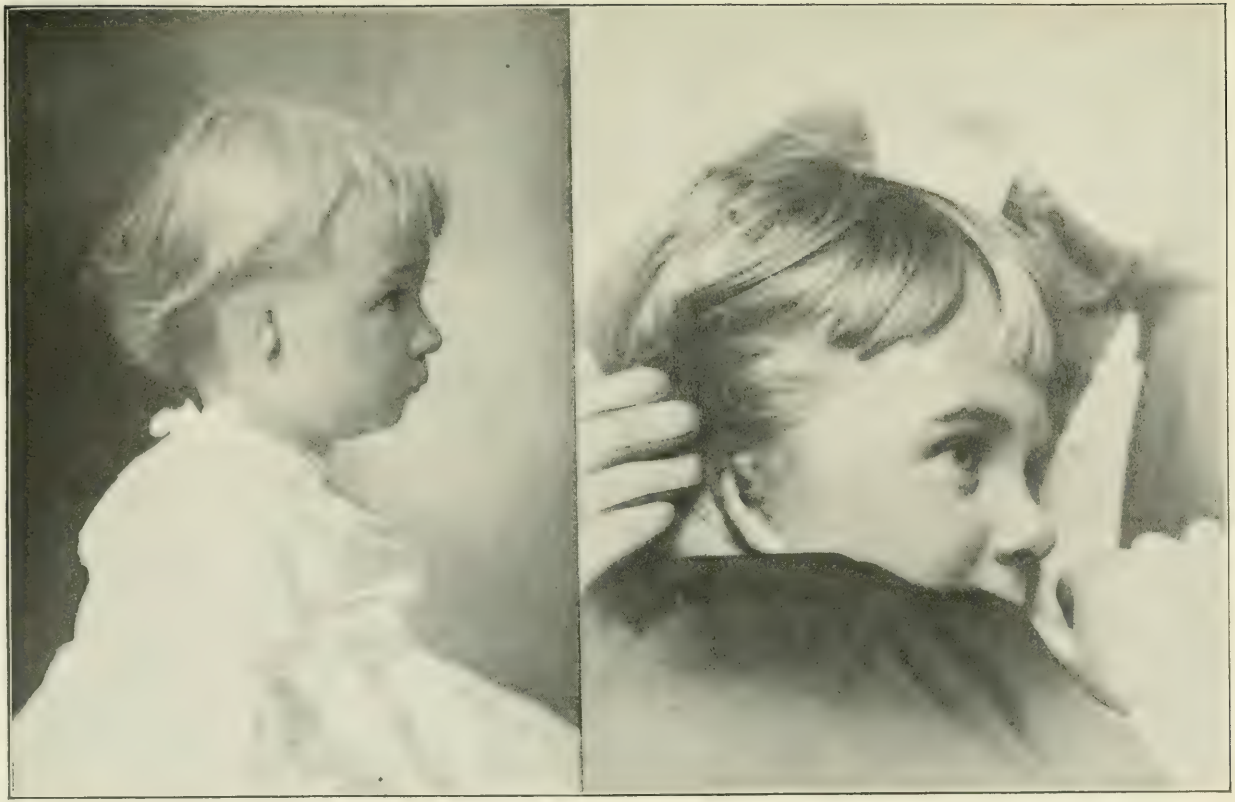
Dr. Duel's case, a girl twelve years of age with bilateral congenital microtia with total osseous atresia, was able before

operation to hear loud sounds, and had been taught to write, read lips and speak a few words indistinctly. She was operated on through the mastoids on both sides, the openings being made far back intentionally in order to avoid entering a possibly normal middle ear cavity. A fairly large antrum and aditus was found leading to a tympanum of moderate size; ossicles were apparently present. As the operation did not extend beyond the antrum, and aditus, definite knowledge of the tympanic cavities was not obtained. Following the procedure suggested by Michael Jaeger,[9] as early as 1837, the operation was limited to this extent, the cavities were skin grafted, and there was marked improvement in the hearing for a short time. As the case was afterwards placed in my hands for dressing I have to assume responsibility for its ultimate failure, for the grafts did not take, and I was utterly unable to keep patulous an opening down to the aditus. There was no permanent improvement of hearing in this case.

Page's case: Another case,[10] one of bilateral congenital microtia with atresia now five years of age has been under my observation since he was seven weeks old. Tuning fork and bone conduction tests were of no use, but as whistling on one's fingers easily demonstrated the presence of hearing, as did tests with the voice, his right ear was operated on when he was two years of age.

The X-ray revealed an antrum and tympanum with what looked to be ossicles present on both sides, and mastoids of fair size.

The usual curved incision was made behind the rudimentary auricle, and when that was displaced forward no evidence of bony meatus was found except a barely noticeable depression, or rather unevenness, in the bone at the point where the meatus might have been expected to be. The cortex of the mastoid was removed and every cell beneath it was found filled with thick muco-purulent fluid 'though there was no external sign to indicate the presence of such a condition, the child's temperature being normal. A fairly large antrum was found and from this a small aditus leading to a contracted middle ear. All were filled with the same muco-puru-



Photograph taken shortly before first operation.

Photograph taken two years after operation on right ear.

Right ear operated on January 20, 1911.

Photograph taken shortly before first operation.

Photograph taken two months and six days after operation on left ear.

Left ear operated on October 16, 1912.

lent fluid. Experience with Dr. Duel's case impressed me with the difficulty of keeping open a canal down to the aditus so I determined to invade the tympanum and get as large an exposure of its internal wall as possible. The bone corresponding to the location of the meatus was cut down to the aditus, making a large single cavity in the bone. No evidence of fibrous canal, or drum-membrane, or annulus was found. In removing the bone from along the line of what should have been a bony canal, a hard tubular line of bone was encountered external to the horizontal semi-circular canal, and it was at first thought to be the aqueductus falopii, despite its faulty position. More bone was removed, however, and it was found to be the edge of a rudimentary incus which was imbedded in the bone. No evidence of a membrane, or of the other two ossicles was discovered. I am confident, however, that a stapes was present, although I was unable to definitely see it on account of the bleeding, and the overhanging facial ridge which prudence forbade my lowering to the extent it might have been lowered in a less abnormal case. The middle ear was a narrow, slit-like cavity from without inward, but fairly wide from the orifice of the eustachian tube back to the aditus and from above downward. The eustachian tube was patent and the tympanum was lined with membrane continuous with that of the tube, and the whole cavity was filled with the same muco-purulent fluid that was in the antrum and cortical cells. A large meatus nearly two centimeters in diameter was cut in the rudimentary auricle, the skin being turned in as far as possible in order to retain it, as there was no cartilage to give it support. The posterior incision was closed and the cavity was skin grafted ten days later. A caloric test after the operation was markedly positive within a few seconds.

Two years later the case was presented before the Otological section at the New York Academy of Medicine.[10] The meatus was then about 5 millimeters in diameter and easily admitted a speculum of moderate size without stretching. The interior of the cavity was clean, white and dry—a medium-sized radical cavity.

Because of the easily apparent improvement in the child's hearing the mother was anxious to have his other ear operated on, as he was then becoming more attentive and had begun to form a few short sentences of simple words. The concensus of opinion at the meeting on this occasion was that the second operation was decidedly warranted.

The same procedure was followed in the second as in the first operation. Examination revealed no evidence of a meatus, or a bony canal. No pus was found in the cells, as there had been on the opposite side. An annulus-tympanicus was found and what appeared to be an indefinite web-like drum-membrane, also a poorly formed malleus. The tympanum was itself wider than the one on the opposite side and contained in addition to the undeveloped malleus a well formed incus and, as was afterwards demonstrated, a stapes.

In forming the meatus a large crucial incision was made, the four flaps of skin were dissected up and reflected, and a very large opening in the soft parts was formed. The flaps were then drawn well in over the edges of the newly constructed meatus, and held in position with sutures. The posterior wound was sutured throughout, and ten days later the interior of the cavity was grafted with Thiersch grafts. Caloric test performed later gave a positive reaction within a few seconds.

His mother, in a letter dated April 24th, 1914, (three years after the first operation, and a year and a half after the second,) states that she speaks to him in the same natural tone of voice she uses for the other children, and he answers promptly. When she calls to him from an adjoining room he replies readily. He imitates different animals, and called her attention a short time ago to the croaking of frogs some distance off. Is bright and talks remarkably well.

The comparison of this case with that of Brühl's shows distinctly the advantage to be gained from operation.

Dahmer's case: Dahmer[11] reports a case of operation on a three-year-old child with bilateral microtia and atresia. He established on each side a bony canal to the tympanic cavities. The tympanic membrane was lacking but the

stapes was present on both sides. Hearing was much improved after operation.

Morris' case: Morris'[12] case is of interest not only on its own account but because it shows that the operator's respect for the general opinion expressed in literature led him to abandon the operation when the completion of it as a "Radical" would very likely have resulted favorably. Stated briefly it was as follows: Boy 10 years of age—Bilateral Rudiments of Auricles, no meati—Facial expression bright and intelligent—Could recognize name and certain commands when spoken in a loud voice by his father—Could utter a few words indistinctly. Testing with tuning forks by air and bone-conduction was unsatisfactory. Attempt was made to follow Jaeger's suggestion of opening antrum as in mastoid operation and keeping wound open. Operation revealed no evidence of meatus, or canal. Cortex was removed where canal should have been. Dura was exposed above, jaw anteriorly, and sinus behind. The last was injured. The other side was not examined. He concludes his report by saying "I offer this not because it teaches much, but because such cases are rare and therefore of interest and because it *emphasizes the fact already well established* that these poor unfortunates are usually beyond surgical, or any other means of relief."

It is not to be denied that such an *opinion* is well established, but that the so-called *fact* is not it is the object of this paper to demonstrate.

Braun's case, not yet published, was similar to this one as was Alexander's, in that the jaw encroached on the line of the canal and overlapped the tympanum to some extent. In both of these cases the improvement was marked after operation. I understand from the former that in his removal of bone forward from the mastoid cavity he exposed the maxillary joint encased in fibrous tissue, and found internal and somewhat posterior to it an annulus, but no drum-membrane. Ossicles were demonstrated in what was practically a normal tympanum on both sides.

Cheatle's case: Cheatle's case,[13] which involved one side only, showed under X-ray mastoid cells and a middle ear tract. No meatus was found on operation and the jaw appeared to articulate with the posterior meatal wall.

Berens' case: Berens' case, not yet reported, in a child five years of age, with double microtia and total osseous atresia also showed on both sides the articulation of the jaw with the anterior wall of the mastoid. This, however, did not interfere with his operation in that he performed what was practically a radical on both sides, finding no drum-membrane on either side, but rudimentary malleus and incus on one and rudimentary malleus on the other. Stapes was present on both sides. His cavity was dermatized with the aid of skin flaps taken from the region of the wound and turned in, lining it completely.

Dr. Berens tells me that before the operation the child was stupid and dumb, and that within an astonishingly short time her whole facial expression changed and she began to acquire speech.

It will be seen from these cases cited that in the majority of them the jaw articulates with what is practically the posterior meatal wall or anterior border of the mastoid, as it is invariably the anterior wall of the canal that is lacking. This was true in my own case, but not perhaps to the extent that it was in some of the others, as it did not interfere at all with my exposure of the internal wall of the tympanum.

Alexander[6] thinks that the cause of failure in operating on such cases has been due to the fact that the attempt is usually made to operate along the line of the canal, and as the jaw almost invariably encroaches on this line there is therefore no room to make a bony canal. It is because of this, he thinks, that the general prejudice against operation has been formed. The procedure suggested by Michael Jaeger in 1836, already referred to, obviates this fault, and to this extent it is valuable. Swartze,[14] writing in 1885, referring to it, states that as far as he knows the operation had not been attempted.

Cassel[5] after remarking on the surgical importance of bearing in mind the process of development of the organ of hearing in these cases, states that "If, notwithstanding the possibility of a normal labyrinth, in any given case, we find on examination that the eustachian tube is malformed beyond the possibility of executing its function, or totally absent, any attempt at remedying the malformation of the external ear is hardly justifiable inasmuch as the tympanum and it contents may be, and probably are, involved in the abnormal change of form. With a normal eustachian tube, however, any such attempt is not only not questionable but perfectly warranted."

Alexander[6] makes practically the same statement, for he says those cases with the patulous eustachian tube and a rudimentary pitting in the region of the bony canal are most favorable for operation, but those in which the tube is closed have such marked deformity of the middle ear he thinks little improvement can be gained for them by operation.

My own contention is that even in these cases which they exclude a stapes usually exists with a normal labyrinth, and that being the case a wide meatus and cavity exposing a thin lining of skin over the oval window and stapes can be obtained with marked improvement of hearing.

It is then simply a matter of exposing and keeping patulous a canal down to the internal wall of the tympanum, which invariably contains a stapes, and is not involved in the malformation, since its formation is from an entirely different origin.

Except in some of the bibliography attached the general attitude in literature will be found to be against operation for the three reasons mentioned, the first of which is, I think, shown to be unjustified by the development of the temporal bone, the second by the substitution of simpler and more practical tests for hearing, and the third requires only an allowance for cicatricial contraction, and careful operative technique and after treatment to refute it.

The conclusions reached are: That operation is warranted in practically every case of congenital bilateral microtia with total osseous atresia which occurs in *an otherwise well formed infant;* that when the child has reached the age of two years and his attention can be attracted by sounds through the air, such as whistles, calls, etc., X-ray photographs should be made (under anesthesia), and operation should be performed through the mastoid posterior to the line of the canal. The antrum should be opened and, unless a normal drum-membrane is found, the contents of the tympanum with the exception of the stapes should be entirely removed and a large exposure of the inner tympanic wall obtained. Injury to the jaw can be avoided by working from the antrum forward. The eustachian tube, if present, should be closed by curetting. The secret of success depends on the allowance for cicatricial contraction in the soft parts and the construction of a large "radical" cavity in the bone. It is of the utmost importance to control the formation of thick connective tissue in the cavity, so within a week or ten days it should be grafted, and if for any reason failure of the grafts should occur, a second or even third attempt to obtain rapid dermatization should be made without delay. The possibility of affording the child improvement of hearing at the critical period necessary for the easy development of speech warrants the early operation.

BIBLIOGRAPHY.

1. Imhofer, R.—Beiderseitige angeborene Gehörgangsatresie. Beitr. z. Anat. Physiol. Path. u. Therap. d. Ohres 4:62 (1910).
2. Politzer, A.—Diseases of the Ear. (1902) p. 815.
3. Kerrison, Philip D.—Diseases of the Ear. (1913) p. 4—6 et al.
4. Castex, A. et Berruyer, G.—Les Malformations de l'Oreille. Bull. d'Oto-Rhinolaryngol. 14:10 (1911).
5. Cassels—(Glasgow Med. Journ., 8:125, 1876).
6. Alexander, G.—Zur chirurgischen Behandlung der kongenitalen Atresie. Zeitschr. f. Ohrenheilk. 55:144 (1908).
7. Politzer, A.—Diseases of the ear. (1902). p. 815.
8. Blumenthal, A.—Zur operation der Mikrotie mit kongenitaler Gehörgangsatresie. Zeitschr. f. Ohrenh. 59:368, (1909).

9. Jaeger, M.—Klinische Beobachtungen über Augen-und Ohrenkrankheiten. Zeitschr. f. Ophthal. 5:1, (1837).
10. —Page, J. R.—"Case of Congenital Bilateral Microtia with Total Osseous Atresia of Both External Auditory Canals." Annals of Otology, Rhinology & Laryngology, Dec., 1912.
11. Dahmer—Zur Missbildung des Gehörganges. Klin. Therap. Wochenschr. 17:1,312, (1910).
12. Morris, S. M.—Microtia. Texas State Jour. Med. 7:14, (1911-12).
13. Cheatle, A. H.—Case of Congenital Malformations of the Auricle and Meatus. Royal Soc. Med. Proc. 4: Otol. Sect. 25, (1910-11).
14. Schwartze, H.—Lehrbuch der Chirurgischen Krankheiten des Ohres. Stuttgart, 1885.

Discussion.

Dr. T. Passmore Berens, New York, said with reference to one of his cases mentioned in the paper: On one side there was what might be described as a rudimentary tragus, on the other side there was an exceedingly small auricle. There was no vestige of an external auditory canal on either side. The child apparently was entirely deaf, for there was absolutely no change in facial expression following any kind of a noise. Even with the tuning forks placed on the vertex there was no apparent response.

In a few months the child was beginning to talk with the other children in the ward and the casual obsever would not notice that the child had trouble with its hearing. It has passed from absolute dumbness to average intelligence. The facial paralysis which was complete on one side cleared up after a few months' massage. Dr. Berens's one idea in operating was not to make what might be called a deep canal, but he endeavored, and successfully, to make what might be called a saucer-wound with the middle ear in the bottom of the saucer.

Dr. B. Alexander Randall, Philadelphia, had seen such good hearing come, in cases of this kind, by way of a patulous Eustachian tube, that he would urge securing a patulous condition of the tube in all these patients. Being rather a stickler for phrases, he would call attention to Dr. Berens'

use of the term "middle ear," as if the antrum were not the middle ear. He thought he heard Dr. Page, in the paper, refer to the development of the external auditory meatus from the first branchial cleft. He would remind him that the first branchial cleft was lachrymal and the second, if persistent, the onomdous aural fistula. The external auditory canal was a different matter, a later-formed involution of the epiblast.

The cases in which he had operated had given him very satisfactory results, but unless he had been able to maintain the patulous condition of the canal he had had no such brilliant results.

In cases of cicatricial stenosis he had used rubber tubing, much as dentists employ it for wedging teeth, in maintaining patulous condition of the canal. A thin sheet of "tea-chest lead" might be used advantageously for the same purpose. It could be rolled in a scroll and distended more and more to meet the needs, yet being rigid enough to resist tissue-contraction.

REPORT OF CASES OF AURAL INFECTION WITH THE STREPTOCOCCUS CAPSULATUS.

By CHARLES E. PERKINS, M. D., New York, N. Y.

I desire to report briefly a few cases of aural infection with the streptococcus capsulatus, or bacillus mucosus, which illustrate some of the forms that infection with this germ may assume.

Case I. An instance of an apparently severe form of infection which cleared up by free drainage through the drum membrane.

M. E., age 33, came under my care April 14, 1912. One week previously, while bathing in a pool, he was nearly drowned and was resuscitated with some difficulty. This was a common pool, about ten feet square, and one may presuppose the presence of germs in abundance. On the following day, he had pain in the left ear, which became severe and was followed in a few days by pain behind the ear.

On examination, he had marked tenderness; the drum membrane was bulging, thickened, and without lustre. A free incision was made. During the next two days, the discharge was profuse and the mastoid tenderness diminished, but pain in the opposite ear developed, with bulging membrane. This was freely incised under gas, and as the first ear was not draining properly, it was re-incised. Four days later, re-incision of the right or second ear involved, became necessary, as it was not draining satisfactorily,—as shown by inspection of canal, increase of mastoid tenderness, and the presence of some spontaneous pain. After this, the case progressed satisfactorily, and the patient was discharged on the eighth day. I saw him, however, at times during the following three weeks, when recovery seemed complete. I have lately examined this patient, after two years, and he has remained well.

During the last three years there have been quite a number of patients with this infection in Dr. Dench's Clinic at the New York Eye and Ear Infirmary that I have treated and that have cleared up without operation. In some, no special mastoid symptoms have been present at any time. Simply being suspicious of the character of the discharge, I have had a smear examined and having found the prevailing germ to be the streptococcus capsulatus, I have kept them under close observation and they have cleared up without mastoid symptoms. Some of them I have seen for two or three months and am satisfied that their recovery was complete. I regret that I cannot give x-ray findings in these cases, but most of them occurred before we were accustomed to place as much reliance in x-ray plates as we do now. I have seen a number of cases of mastoid involvement which at no time were accompanied with aural discharge.

Case II. S. L., female, aged 25, referred by her physician on July 27, 1909; for three months had a small swelling behind the left ear, which appeared just subsequent to confinement. Examination showed a hard fluctuating mass behind and above the ear, about an inch in diameter. The auricle seemed to stand away from the head. The external auditory canal, the membrana tympani, and fundus appeared absolutely normal. I advised postauricular exploration, and at the Infirmary I operated on the following day and found the mastoid completely broken down, a large perisinus abscess, and a large quantity of pus. The tympanic vault was completely walled off by firmly organized tissue, which I did not disturb. Examination of the pus showed infection to be streptococcus capsulatus. The patient made a perfect recovery. At no time was there the slightest discharge from the canal. The wound was completely healed in five weeks.

Case III. Mrs. E., age 62, consulted me on September 18, 1913. Began with pain in left ear, following a cold in preceding March, i. e., six months previously, during which time she had suffered more or less pain in the ear and head. She

was treated by the family physician in all manner of ways except by incision of drum.

Examination showed some loss of lustre and thickening of the membrana tympani, and also slight narrowing of the canal, with mastoid tenderness and thickening of post-auricular tissues. Two and one-half per cent. of sugar in the urine; no acetone.

X-ray examination by Dr. Dixon: Large pneumatic type; septa between cells blurred. Frankly an operative case.

On account of the marked glycosuria, operation was done as rapidly as possible,—total time, 23 minutes. The mastoid was found to be totally disorganized. Large perisinus abscess with immense granulations springing from its walls: pus, abundant, showed infection to be streptococcus capsulatus. Wound healed in six weeks; remains well.

Case IV. M. H., age 65, referred by his physician (on December 20, 1913) who had treated him for about four weeks for tinnitus and impaired hearing in the left ear. There was no pain or mastoid tenderness, but the drum was thickened and without lustre. Inflation produced moist râles.

X-ray by Dr. Dixon: Right, clear pneumatic mastoid; left, same type, but cloudy throughout.

It was not until a week later that tenderness about one inch posterior to the ear developed and he began to have pain at night, then he consented to operation, which showed the mastoid to be disorganized and filled with a large granulating mass springing from the sinus. Large tip cells filled with pus, and an epidural abscess extending fully an inch posterior to the sinus in the cerebellar fossa. Convalescence uneventful; wound healed in five and a half weeks.

Case V. Mrs. M. S., age 65, consulted me on March 2, 1914, with a history of pain in the left ear for seven weeks. Four weeks previously, incision of membrane by a very competent man, followed by no discharge of pus. The mastoid was tender and the overlying tissues were somewhat

edematous. The drum membrane was thickened and lustreless. There was no discharge from the canal. The operation showed the mastoid cells to be disorganized, with epidural abscess at sinus knee. Convalescence slow.

The following case is one of many observed in which the discharge was profuse, the bacteriological examination leading, in a way, to the operation.

Case VI. A. G., male, age 58, came under my care February 12, 1914. Pain in right ear for two weeks. He was treated by his physician, but without incision of the drum. He had marked mastoid tenderness. The membrane, which was thickened, dull red, and lustreless, was incised. The following week, the mastoid tenderness became very much less,—indeed, nearly absent. The discharge, however, continued profuse.

X-ray by Dr. Dixon showed large-celled pneumatic bone, cloudy throughout. Operation showed mastoid thoroughly infiltrated with pus. The wound healed in seven weeks.

The following case was one in which the ear infection seemed very benign.

Case VII. Mr. S., age 45. Has had discharge from right ear for past two winters, lasting each time about one week. About the 1st of January, 1914, he developed an acute inflammation of the middle ear with spontaneous rupture of the membrane, which was incised two days later. The discharge was not profuse at any time. My impression is that I had a smear made and that it showed some benign infection, but I am unable to verify this, as the records are silent on this point. At any rate, the discharge had none of the characteristics which we are accustomed to associate with that due to streptococcus capsulatus. There developed marked spontaneous pain and tenderness in the occiputal and pre-auricular regions, but as there was no mastoid tenderness and the patient was a very nervous individual, this was attributed to neuralgia and hyperasthesia. Nevertheless, on January 16, an x-ray was made by Dr. Dixon: "Left mastoid seems diploetic; right (involved side), indistinct pneumatic; has

appearance of sclerosed bone." One would hardly operate on this x-ray finding alone.

During February the discharge ceased, although inflation always showed fluid in the middle ear, and the membrana tympani remained dull and lustreless. Early in March, a moderate sagging of the superior wall occurred, extending from Shrapnell's membrane outwards, about one-third the length of the canal. The pain and tenderness in the occiput and pre-auricular regions had become very slight, but a tender spot had developed about an inch posterior to the ear.

X-ray examination by Dr. Dixon showed (March 13th): Right mastoid, cloudy. Perisinus abscess has developed since January 16 (date of former examination).

(Note: The beginning of the abscess was shown on former plate, but at the time was so small that it escaped notice).

Operation on March 20. Cortex, sclerosed, but after it was removed pus swelled up and perisinus abscess was found. In this location,—one inch posterior to ear, site of tender spot and abscess shown in x-ray plate, the bone was eroded nearly to the surface by a large granulation springing from the sinus. The patient made an uneventful recovery. The mastoid healed completely in four and a half weeks.

That one is not through with these cases at times, even when an operation has been performed and convalescence is apparently well established, is illustrated by the following case, occurring in the practice of Dr. N. R. Dann, to whose kindness I am indebted for the report.

Case VIII. W. K., age 38, seen on December 4, 1913, in consultation with family physician. Aural suppuration for three weeks. Examination showed profuse aural discharge, decided sagging of canal wall, and extreme mastoid tenderness. Operation same day. Two cortical perforations; pneumatic bone infiltrated with pus; sinus exposed. Wound practically healed on March 15th, i. e., over three months later,—a small sinus, however, persisting. On March 25th, Dr. Dann saw the patient in consultation. He had suddenly gone into collapse. He had severe headache for three days

and nights previously, and had had severe vomiting. Temperature 102.4°. Patient unconscious and picking at the bedclothes. Pupils unequal and do not react to light. Kernig marked. No lumbar puncture; no autopsy.

I think we are justified in concluding:

(1) That this germ has a special affinity for bone tissue.

(2) That in these aural infections, if the passage between the middle ear and the mastoid is large, drainage through the ear may effect a cure, so the importance of early and free incision of the drum membrane cannot be over-estimated.

(3) That the x-ray examination is of great help in these cases.

(4) That an early and free operation is indicated as soon as we are convinced of irreparable mastoid involvement.

(5) That these cases, whether operated or not, should be watched closely throughout, as late complications are more frequent, perhaps, than in other infections.

(6) It would seem that patients advanced in life, and diabetics, are more liable to this infection than to other forms.

Discussion.

Dr. E. B. Dench, New York City, thought all recognized the severity of streptococcus capsulatus infection, but all did not recognize the fact that a good many of these cases got well without mastoid operations. He had reported thirteen cases in private practice, only two of which came to operation. If seen early, and the drum membrane incised before there were complications, operation would not be necessary. While a smear might show streptococcus capsulatus infection, he did not think it necessary to operate upon the mastoid in all cases. He cited a case in which there had been two previous attacks of middle ear infection. With the third attack he incised the drum membrane and found the streptococcus capsulates. In order to be absolutely sure, he had an X-ray plate made three days later. There was very

little discharge. The mastoid was cloudy. The patient made a perfect recovery about five weeks after the incision of the drum membrane. When trying to account for the cloudy mastoid he remembered the two previous attacks of otitis media, which probably explained it.

THE RELATION OF THE LABYRINTH TO THE CEREBELLUM AND THE CEREBRUM.

(A Lantern Demonstration of Photographs.)

(On account of trouble in Europe the plates could not be obtained in time for publication in the Transactions, but will be published later).

By J. GORDON WILSON and F. H. PIKE.

(Presented by J. Gordon Wilson.)

Some years ago Dr. Pike and I became convinced that in the labyrinth of the ear we have a peripheral sense organ which is capable of helping towards a solution of some of the present mysteries of the central nervous system. There is here the peripheral ending of a nerve pathway of wider extension and of wider associations than the central optic path of which the ophthalmologist has made so much use. It has long been recognized that this pathway could be excited in a variety of ways, experimentally as well as in disease. We felt justified therefore in believing that cranial lesions in the course of its ramifications and associations would modify the results of peripheral labyrinthine stimulations.

About six years ago we began an investigation, the objects of which may be thus briefly summarized:—

(1) To study the effect of stimulation of the end organ of the vestibular nerve.

(2) To study the immediate and the remote results of destruction of the labyrinth or of the eighth nerve on one or on both sides.

(3) To observe the effect of removal of various parts of the brain on phenomena which have been observed to follow stimulation or destruction of the labyrinth.

(4) To investigate the paths of connection between the labyrinth and the eye muscles—the vestibular-optic path.

(5) To apply the results of this experimental work to a consideration of the phenomena of nystagmus, vertigo, and the sensation of rotation.

We need not insist before this audience that such an investigation could be carried out in animals where we can control the extent of the lesion and where there are no septic disturbances to befog our observations.

There are to be distinguished two varieties of nystagmus.

(1) Nystagmus (oscillatory nystagmus) a movement of the eyes in all directions from the central point of rest.

(2) Nystagmus twitching (alternating nystagmus) when the movement is quicker in one direction.

Labyrinthine nystagmus belongs to the latter group. It is well here to emphasize the fact that labyrinthine nystagmus consists of two distinct though associated movements viz, a slow deviation followed by a quick return to the primary position. The following definite reasons have led to the separation of these two movements in regard to cause and source:—

(1) The slow deviation alone occurs in the earlier stages of an anesthetic. The quick movement only appears as the effect of the anesthetic wears off, and the animal regains consciousness.

(2) When the anesthetic is pushed, the slow deviation is one of the last of the cranial reflexes to disappear. It persists nearly or quite as long as the corneal reflex.

(3) Bartels has shown that the slow movement alone can be produced by rotation in sleeping children, and that in prematurely born children (seven months) the slow deviation alone can be elicited for some time after birth.

(4) We have shown in the previous paper (Wilson and Pike, Phil. Trans. Roy. Soc., 1912, p. 127-160) that the quick movement can be eliminated by destruction of the cerebrum and thalamus while the slow persists. As there pointed out, it appears to us that the paths along which the slow deviation occurs, are fairly definitely established.

As a preliminary it was necessary to ascertain the effect of stimulation of one and then of both labyrinths. Though many investigators had studied this, yet it appeared to us that many points must be more clearly defined. We soon

found that the position of the animal affected our results and so our experiments were all carried out with the animal in a definite position and through a definite opening. The eye movements gave so marked a reaction that we felt that we must carefully observe and compare such deviations before we could contrast the labyrinthine deviation with cerebral and cerebellar ocular deviations.

The operative measures employed to expose or destroy the labyrinth have been fully detailed elsewhere (Wilson and Pike, loc cit). It was developed independently by Camis and by us at about the same time. It is sufficient here to state that it consists in exposing the otic bone through a trephine opening in the pars mastoidea of the temporal bone just posterior to the angle at which the zygomatic process passes anterior from the linea nuchae superior.

The forms of stimulation used were:

1. Mechanical or physical.
2. Electrical—constant current.

TABLE OF RESULTS.
(Dog.)

Stimulation of Left Labyrinth through Trephine Wound.	Left Eye (deviation).	Right Eye (deviation).
Cold water	To left and up	To left and up
Hot water	To right and down	To right and down
Electrical, anode; on	To left and down	To left and up
Electrical, cathode; on	To right and up	To right and down
Pressure	To right and up	To right and up
Stimulation of Right Labyrinth through Trephine Wound.	**Left Eye (deviation).**	**Right Eye (deviation).**
Cold water	To right and up	To right and up
Hot water	To left and down	To left and down
Electrical, anode; on	To right and down	To right and down
Electrical, cathode; on	To left and down	To left and up

Stimulation of Both Labyrinths Simultaneously.		Left Eye (deviation)	Right Eye (deviation)
Left Ear	*Right Ear*		
Cold water	Cold water	Up	Up
Hot water	Hot water	Down	Down
Electrical, anode; on	Electrical, anode	No result	No result
Electrical, cathode; on	Electrical, cathode	No result	No result
Electrical, cathode; on	Electrical, anode	To right, down	To right, down
Electrical, anode; on	Electrical, cathode	To left, down	To left, down

Summary of Results:—

(1) Under an anesthetic a dog shows only a deviation of the eyes, varying with the labyrinth stimulated and with the stimulus employed.

(2) (a) Hot water and the negative pole give the same deviation of the eyes.

(b) Cold water and the positive pole give the same deviation of the eyes.

(c) The deviation produced by the former (a) is in the opposite direction to that of the latter (b).

(3) In electrical stimulation of both labyrinths simultaneously, equal electrical stimulation of both sides gives no results. Any inequality of stimulation on the two sides is attended with eye movements away from the side on which the stronger stimulus is applied. The cathode is a stronger stimullatus than the anode. Therefore when the cathode is over the left otic bone, as when the left labyrith only is stimulated with the negative pole, both eyes move to the right.

Figures 3 to 11 and the figures of cats show the results of destruction of one labyrinth or of both labyrinths, on the same day or at different dates. The photographs it will be noted have been taken at different dates.

General Summary of Results—after destruction of one or of both labyrinths:—

A. Deviation of the Eyes and Nystagmus.—Immediately after unilateral destruction of the labyrinth the eye of the

injured side is deviated down; later, down and slightly out. On the injured side the deviation is towards the side of lesion, occasionally with a slight upward movement. Later, the deviation is down and to the injured side in both eyes. Speaking generally, it may be said that after labyrinthine destruction the eyes are deviated to the side of the lesion.

The nystagmus is composed of two distinct phases, a slow movement and a quick jerk back. The nystagmus is synchronous in both eyes, and is constantly present for some days following the destruction. The irregularity of the direction of the movements of the two eyes within the first 24 hours is marked. When the animal comes out of the anesthetic it is nearly vertical in the eye of the operated side and horizontal in the eye of the non-operated side. The variation appears to us to be due to the resultant (a) of the relative over-activity of the opposite labyrinth, and (b) the irritation of the vestibular nerve in the wound, and, it may be, the canal most affected.

B. The General Attitude.—Immediately after the operation the attitude of the animal is very characteristic. After allowing the animal to come out from under the influence of the anesthetic, if it be placed on the floor ventral side downward so that the hind feet are squarely on the floor, there is a torsion of the head to the injured side so that relative to the dorsoventral axis of the body it is inclined about 30°—45° from the vertical at the shoulders. The injured side of the head lies towards the floor. The head is twisted about 70° to the injured side. On attempting to move, the animal rolls over towards the injured side. The animal is unable to walk, and, if stood up on its feet, will fall over to the injured side.

On succeeding days the animal becomes more steady on its feet, until at the end of a week it seldom falls to one side, and walks in a straight line. At the end of the second week the animal has assumed its permanent attitude. It now stands erect with torsion of the head to one side, always with the occiput of the injured side twisted downward and forward, and the anterior part of the body inclined somewhat to the injured side.

The effect of removal of the second labyrinth varies somewhat with the length of time after the first operation, but, in general, the effects are less severe the longer the interval between the operations. The head and the anterior part of the body are not twisted to the recently injured side; on the contrary, the head comes nearly but not quite back to its normal position and remains there. The nystagmus is present for a few hours but has disappeared by the next day.

Having defined the reactions which follow irritation and destruction of the labyrinth, it becomes necessary to ascertain what influence the cerebellum has on those results. In order to do so the cerebellum was removed partly or entirely, and at a later date the labyrinth was stimulated and destroyed.

The results are given very briefly in the following table, where they are contrasted with labyrinthine removal on the same side. The eye movements may be specially referred to. While the deviation of the eyes in cerebellar lesions, best observed if the dog be at rest, is usually to the right, yet this deviation is inconsistent and varies considerably in the two eyes. The day after the operation it may be to the left in the left eye (operated side) and normal in the right. But the oscillatory movements are distinctly and consistently different in cerebellar and in labyrinthine lesions.

	Left Half of Cerebellum	Left Labyrinth
Deviation of eyes	To right	To left
Movement of eyes	Quick oscillations in any plane, especially when disturbed or when an object is looked at	Slow movement to left with quick return to right (Labyrinthine nystagmus)
	If head is turned 90° to left oscillations are increased	If head is turned 90° to left, nystagmus is diminished
	No twitching of M. corrugator supercilii medialis	Marked twitching of M. corrugator supercilii medialis
Attitude	Left pleurosthotonus	Torsion of head so that occiput is approximated to left shoulder
	Snout to left	Snout to right
	Prefers to lie on right side	Prefers to lie on left side
Part affected	Posterior chiefly	Anterior chiefly

The Effect of Lesions of Cerebrum on Labyrinthine Reactions:—

Removal of a part or of the whole of the cerebellum made it obvious that the deviation of the eyes and the nystagmus resulting from labyrinthine stimulation or destruction was little if at all influenced directly from the cerebellum, and further that the pathway to the eye nuclei lay outside the cerebellum. It therefore became necessary to ascertain—

(1) Whether the path from the labyrinth to the eye nuclei is direct by the fasciculus longitudinals medialis (posterior longitudinal bundle) or whether other higher centres are involved.

(2) Whether removal of various parts of the cortex of the cerebrum changes the labyrinthine effects. Since the quick component in the labyrinthine nystagmus could occur and could be produced only in the conscious state, suggesting a cerebral origin, it was determined to ascertain—

(3) From what portion of the cerebrum the quick component comes.

To accomplish this it was necessary to remove various parts of the cerebrum, and either immediately after or at a later date to observe the effects of labyrinthine stimulation or destruction.

The following as well as other experiments were done:

1. Destruction of both occipital lobes of the cerebrum.
2. Destruction of approximately half of the cerebral cortex.
3. Removal of entire cerebral cortex.
4. Complete removal of whole cerebrum with severance of corpora quadrigemina and splitting of cerebellum in the median line.
5. Effect of labyrinthine stimulation and destruction on decerebrated animal.

The results of these experiments may be thus summarized:—

Normal results from stimulation of the labyrinth, i. e. the deviation of the eyes can be obtained when the whole cerebrum and thalami are removed, leaving nothing but pons,

medulla, and their connections with the cranial nerves. The ocular movements in labyrinthine stimulation and after labyrinthine destruction are not merely the spasmodic flickering of a muscle, or an inaccurate and impotent movement as of a foot that never hits anything, such as pass for reflex responses in a dog with transacted spinal cord, but full exact movements of normal magnitude.

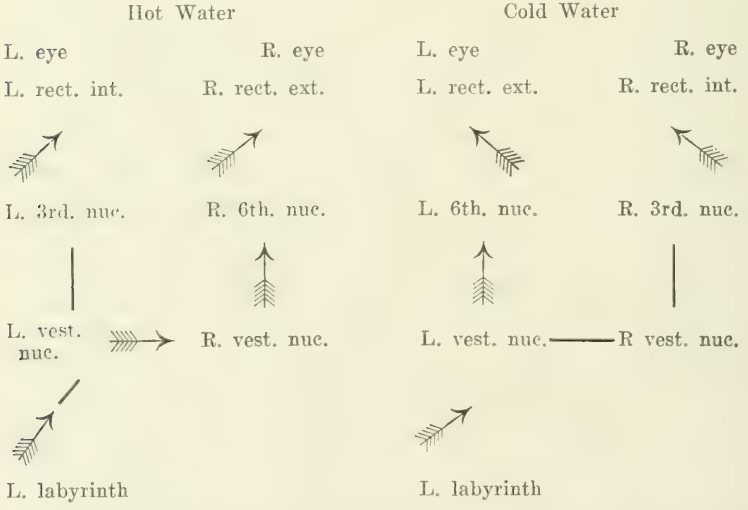
In all vertebrates so far used for experiment, and particularly in mammals, lesions of the membranous labyrinth of the ear are, without exception, attended by torsion of the anterior part of the body, and displacement of the eyes. The torsion of the head is permanent in all these forms except the human. The eye movements alone differ, and these differences are intimately associated with the increasing magnitude and complexity of development of the cerebrum in the vertebrate phylum. The typical results of lesions of the membranous labyrinth, excepting nystagmus, are very largely unaffected by the presence or absence of the phylogenetically newer pathways.

The greater capacity for adjustment following certain lesions or removals of nervous substance, inherent in the enormous development of association tracts in the central nervous system of the higher vertebrate, may tend to obscure or mask, because of the completeness of recovery following labyrinthine extirpation, some of the essential features of such a lesion in the higher forms. But the certainty and the severity of the motor disturbances immediately attending labyrinthine extirpation in the higher mammals is evidence of the persistence of the function of the vestibular mechanism. The more complete recovery in man is not necessarily to be interpreted, as Barany believes, as evidence of the retrogression of the vestibular system in man, but may be regarded as evidence of the much greater degree of adaptability on the part of the highly complex zomatic motor system, a point of view widely at variance with Barany's.

Nystagmus occurring in the dog and cat and monkey as a result of labyrinthine stimulation or destruction, consists of

two phases—a slow deviation followed by a quick return—varying definitely in direction with the labyrinth which is stimulated or destroyed. As we have shown, these phases are distinct. Thus the slow deviation is not affected by removal of the cerebellum, nor is it affected by complete removal of the anterior corpora quadrigemina, and longitudinal splitting of cerebellum. In short, if the anatomical path from the labyrinth through the vestibular nuclei to the eye nuclei (posterior longitudinal bundle) be intact, the deviation can be produced by stimulation, and appears after destruction, so soon as the animal begins to pass out from under the influence of the narcotic. Deviation appears at a time when the reflex responses of the leg muscles are completely in abeyance, and pari passu with the corneal reflex.

The following diagrams illustrate the action of hot or cold water to produce the deviations; the experiments on which they are based will be published later:—



The quick phase of nystagmus, though not affected by the removal of the cerebellum or of the greater part of the occipital cortex, is affected by removal of the whole cerebral

cortex. It does not occur in animals completely decerebrated, including optic thalamus, even though the oculovestibular tract be unimpaired. Further, it appears later than the deviation, and can only be produced, or will occur, when the animal emerges from the effect of the anesthetic.

We have attempted in dogs, cats and monkeys to find some region in a hemisphere where a lesion will modify or abolish the nystagmus which follows stimulation or destruction of the labyrinth. In the course of the experiments we have removed the various areas of the cortex, frontal, parietal, occipital, and temporal, and have also at times destroyed the subcortical ganglionic masses. Having destroyed these areas, we have then stimulated one or both labyrinths with the electrical current, with hot and with cold water, and by rotation. We have also in many of these cases destroyed one labyrinth either concomitantly with or subsequent to the cerebral lesion—a labyrinth either on the side of, or opposite to, the cerebral lesion. We cannot at this time enter into the details of the results, which will be published in full later, but we desire to state here that the only region where we obtain consistent alteration of the labyrinthine simulation, and that only so far as the quick component is concerned, is the region of, or adjacent to, the temporal lobe. In these cases we found that stimulation of the labyrinth on the side opposite the cerebral lesion with cold water or with the anode, gave typical labyrinthine nystagmus lateral in character, while stimulation with hot water or the cathode gave deviation, but no lateral nystagmus. We also found that destruction of the labyrinth on the side of the cerebral lesion gave no nystagmus. There was further noted in some of our cases that the eye on the side of the lesion showed a rotary movement around an anteroposterior axis, while this movement was wanting in the eye of the opposide side.

To sum up, our idea of the mechanism of nystagmus is first an agency somewhere which produces a deviation of the eyes from the primary position of equilibrium or the primary position of the line of fixation of Listing. In labyrinthine nystagmus this agency lies in the labyrinth. The internal

rectus muscle on the side of the slow deviation and the external rectus on the opposite side are subjected to a strain greater than usual. This strain resulting in the stimulation of the sensory or afferent endings of the eye muscles, sets up afferent impulses, which are conveyed to the cerebrum along the afferent fibres in the third, fourth, and sixth cranial nerves; (Tozer and Sherrington, Proc. Roy. Soc. 1910, p. 450). These afferent impulses in their turn set up efferent impulses in the oculomotor cells of the cerebrum, which result in a quick jerky contraction of the internal rectus on the side of the slow deviation, and of the external rectus of the opposite side, with relaxation of the antagonistic muscles. The effect is a restoration of the eyes to the primary position. The eyes do not go beyond this median position when the cerebrum is intact, since the afferent impulses to contraction of the external and internal rectus muscles concerned cease when the eyes reach this position of equilibrium.

Lesions of the cerebral hemisphere on the side of the slow deviation of the eyes abolish or reduce the quick component, since it is from the hemisphere of this side that the efferent impulses concerned in the pulling of the eyes back to the median line arise.

LANTERN SLIDES WHICH WERE THROWN.

1. Field of operation. Fig. Phil. Trans.
   (a) Dog—Trephine opening.
   (b) Dog—Normal skull.
3. Right labyrinth destroyed.
   Dog of April 25, 1909. Photographed May, 1909. Fig. 2. Roy. Soc. Trans.
4. Dog of April 25, 1909. Before second labyrinth destroyed. Photo. March 4, 1911. Fig. 2.
5. Dog of April 25, 1909. After second labyrinth destroyed. Photo. May, 1911.
6. Dog of April 25, 1909. After second labyrinth destroyed. Photo. May, 1911. Phil. Trans. Fig. 9.
7. Dog of April 25, 1909. After second labyrinth destroyed. Photo. May, 1911.

8a. Dog of Jan. 1912.
   (a) Both labyrinths destroyed. Phil. Trans. Fig. 10 and 11.

8b. (b) Both labyrinths destroyed. Photographed Nov. 1912.

9. White rat. Left labyrinth destroyed. Phil. Trans. Fig. 3.
10. White rat. Left labyrinth destroyed. Phil. Trans. Fig. 4.
11. White rat. Both labyrinths destroyed. Phil. Trans. Fig. 12.

12. White rat. Left half of cerebellum removed.
13. White rat. Right cortex of cerebrum removed. Phil. Trans. Fig. 15.
14. Dog of April 24, 1911. Left lateral lobe and posterior part of vermis of cerebellum destroyed.
15. Dog of April 24, 1911. Same dog with left labyrinth destroyed.
16. Dog of May 4, 1911. Complete destruction of cerebellum.
17. Dog of May 4, 1911. Complete destruction of cerebellum. Phil. Trans. Fig. 13.
18. Dog of May 4, 1911.
19. Dog of May 4. After left labyrinth destroyed.
20. Dog of Nov. 3, 1911. Cerebellum and left labyrinth destroyed. Killed Dec. 20. P. M. Removal of vermis, middle lobe, and large part of interior of each lateral lobe.
21.
22a. Dog of Oct. 25, 1911. Right cerebral cortex removed.
23. Dog of Oct. 25, 1911. Left labyrinth destroyed.
24. Dog of Oct. 25, 1911. Left labyrinth destroyed.
25. Dog of Oct. 25, 1911. Left labyrinth destroyed.
26. Dog.
    A. Cerebellum removed.
    B. Cerebrum removed, May 2, 1911.
    C. Normal.
27. Dog.
    A. Cerebrum removed, June 23, 1911.
    B. Half of cerebellum removed.
28. A. Intact brain.
    B. Occipital lobes of cerebrum removed. June 12, 1911.
29. Right cerebral cortex removed. Oct. 25, 1911.

Cats. Right labyrinth removed. Phil. Trans. Fig. 6. 7, 8.

(a) Pigeon after removal of cerebellum.
(b) Pigeon after removal of right labyrinth.
(c) Frog after removal of right labyrinth.
(d) Snakes in water.

[Supplement to the Transactions of the American Otological Society, 1914.]

THE RELATION OF THE LABYRINTH TO THE CEREBELLUM AND THE CEREBRUM.

(A Lantern Demonstration of Photographs.)

(On account of trouble in Europe the plates could not be obtained in time for publication in the Transactions).

By J. GORDON WILSON and F. H. PIKE.

(Presented by J. Gordon Wilson.)

Some years ago Dr. Pike and I became convinced that in the labyrinth of the ear we have a peripheral sense organ which is capable of helping towards a solution of some of the present mysteries of the central nervous system. There is here the peripheral ending of a nerve pathway of wider extension and of wider associations than the central optic path of which the ophthalmologist has made so much use. It has long been recognized that this pathway could be excited in a variety of ways, experimentally as well as in disease. We felt justified therefore in believing that cranial lesions in the course of its ramifications and associations would modify the results of peripheral labyrinthine stimulations.

About six years ago we began an investigation, the objects of which may be thus briefly summarized:—

(1) To study the effect of stimulation of the end organ of the vestibular nerve.

(2) To study the immediate and the remote results of destruction of the labyrinth or of the eighth nerve on one or on both sides.

(3) To observe the effect of removal of various parts of the brain on phenomena which have been observed to follow stimulation or destruction of the labyrinth.

(4) To investigate the paths of connection between the labyrinth and the eye muscles—the vestibular-optic path.

(5) To apply the results of this experimental work to a consideration of the phenomena of nystagmus, vertigo, and the sensation of rotation.

We need not insist before this audience that such an investigation could only be carried out in animals where we can control the extent of the lesion and where there are no septic disturbances to befog our observations.

There are to be distinguished two varieties of nystagmus.

(1) Nystagmus (oscillatory nystagmus) a movement of the eyes in all directions from the central point of rest.

(2) Nystagmus twitching (alternating nystagmus) when the movement is quicker in one direction.

Labyrinthine nystagmus belongs to the latter group. It is well here to emphasize the fact that labyrinthine nystagmus consists of two distinct though associated movements viz, a slow deviation followed by a quick return to the primary position. The following definite reasons have led to the separation of these two movements in regard to cause and source:—

(1) The slow deviation alone occurs in the earlier stages of an anesthetic. The quick movement only appears as the effect of the anesthetic wears off, and the animal regains consciousness.

(2) When the anesthetic is pushed, the slow deviation is one of the last of the cranial reflexes to disappear. It persists nearly or quite as long as the corneal reflex.

(3) Bartels has shown that the slow movement alone can be produced by rotation in sleeping children, and that in prematurely born children (seven months) the slow deviation alone can be elicited for some time after birth.

(4) We have shown in the previous paper (Wilson and Pike, Phil. Trans. Roy. Soc., 1912, p. 127-160) that the quick movement can be eliminated by destruction of the cerebrum and thalamus while the slow persists. As there pointed out, it appears to us that the paths along which the slow deviation occurs, are fairly definitely established.

As a preliminary it was necessary to ascertain the effect of stimulation of one and then of both labyrinths. Though many investigators had studied this, yet it appeared to us that many points must be more clearly defined. We soon

found that the position of the animal affected our results and so our experiments were all carried out with the animal in a definite position and through a definite opening. The eye movements gave so marked a reaction that we felt that we must carefully observe and compare such deviations before we could contrast the labyrinthine deviation with cerebral and cerebellar ocular deviations.

The operative measures employed to expose or destroy the labyrinth have been fully detailed elsewhere (Wilson and Pike, loc cit). It was developed independently by Camis and by us at about the same time. It is sufficient here to state that it consists in exposing the otic bone through a trephine opening in the pars mastoidea of the temporal bone just posterior to the angle at which the zygomatic process passes anterior from the linea nuchae superior.

The forms of stimulation used were:

1. Mechanical or physical.
2. Electrical—constant current.

TABLE OF RESULTS.
(Dog.)

Stimulation of Left Labyrinth through Trephine Wound.	Left Eye (deviation).	Right Eye (deviation).
Cold water	To left and up	To left and up
Hot water	To right and down	To right and down
Electrical, anode; on	To left and down	To left and up
Electrical, cathode; on	To right and up	To right and down
Pressure	To right and up	To right and up
Stimulation of Right Labyrinth through Trephine Wound.	**Left Eye (deviation).**	**Right Eye (deviation).**
Cold water	To right and up	To right and up
Hot water	To left and down	To left and down
Electrical, anode; on	To right and down	To right and down
Electrical, cathode; on	To left and down	To left and up

Stimulation of Both Labyrinths Simultaneously.		Left Eye (deviation)	Right Eye (deviation)
Left Ear	*Right Ear*		
Cold water	Cold water	Up	Up
Hot water	Hot water	Down	Down
Electrical, anode; on	Electrical, anode	No result	No result
Electrical, cathode; on	Electrical, cathode	No result	No result
Electrical, cathode; on	Electrical, anode	To right, down	To right, down
Electrical, anode; on	Electrical, cathode	To left, down	To left, down

Summary of Results:—

(1) Under an anesthetic a dog shows only a deviation of the eyes, varying with the labyrinth stimulated and with the stimulus employed.

(2) (a) Hot water and the negative pole give the same deviation of the eyes.

(b) Cold water and the positive pole give the same deviation of the eyes.

(c) The deviation produced by the former (a) is in the opposite direction to that of the latter (b).

(3) In electrical stimulation of both labyrinths simultaneously, equal electrical stimulation of both sides gives no results. Any inequality of stimulation on the two sides is attended with eye movements away from the side on which the stronger stimulus is applied. The cathode is a stronger stimulus than the anode. Therefore when the cathode is over the left otic bone, as when the left labyrith only is stimulated with the negative pole, both eyes move to the right.

Figures 3 to 11 and the figures of cats show the results of destruction of one labyrinth or of both labyrinths, on the same day or at different dates. The photographs it will be noted have been taken at different dates.

General Summary of Results—after destruction of one or of both labyrinths:—

A. Deviation of the Eyes and Nystagmus.—Immediately after unilateral destruction of the labyrinth the eye of the

injured side is deviated down; later, down and slightly out. On the injured side the deviation is towards the side of lesion, occasionally with a slight upward movement. Later, the deviation is down and to the injured side in both eyes. Speaking generally, it may be said that after labyrinthine destruction the eyes are deviated to the side of the lesion.

The nystagmus is composed of two distinct phases, a slow movement and a quick jerk back. The nystagmus is synchronous in both eyes, and is constantly present for some days following the destruction. The irregularity of the direction of the movements of the two eyes within the first 24 hours is marked. When the animal comes out of the anesthetic it is nearly vertical in the eye of the operated side and horizontal in the eye of the non-operated side. The variation appears to us to be due to the resultant (a) of the relative over-activity of the opposite labyrinth, and (b) the irritation of the vestibular nerve in the wound, and, it may be, the canal most affected.

B. The General Attitude.—Immediately after the operation the attitude of the animal is very characteristic. After allowing the animal to come out from under the influence of the anesthetic, if it be placed on the floor ventral side downward so that the hind feet are squarely on the floor, there is a torsion of the head to the injured side so that relative to the dorsoventral axis of the body it is inclined about 30°—45° from the vertical at the shoulders. The injured side of the head lies towards the floor. The head is twisted about 70° to the injured side. On attempting to move, the animal rolls over towards the injured side. The animal is unable to walk, and, if stood up on its feet, will fall over to the injured side.

On succeeding days the animal becomes more steady on its feet, until at the end of a week it seldom falls to one side, and walks in a straight line. At the end of the second week the animal has assumed its permanent attitude. It now stands erect with torsion of the head to one side, always with the occiput of the injured side twisted downward and forward, and the anterior part of the body inclined somewhat to the injured side.

The effect of removal of the second labyrinth varies somewhat with the length of time after the first operation, but, in general, the effects are less severe the longer the interval between the operations. The head and the anterior part of the body are not twisted to the recently injured side; on the contrary, the head comes nearly but not quite back to its normal position and remains there. The nystagmus is present for a few hours but has disappeared by the next day.

Having defined the reactions which follow irritation and destruction of the labyrinth, it becomes necessary to ascertain what influence the cerebellum has on those results. In order to do so the cerebellum was removed partly or entirely, and at a later date the labyrinth was stimulated and destroyed.

The results are given very briefly in the following table, where they are contrasted with labyrinthine removal on the same side. The eye movements may be specially referred to. While the deviation of the eyes in left cerebellar lesions, best observed if the dog be at rest, is usually to the right, yet this deviation is inconstant and varies considerably in the two eyes. The day after the operation it may be to the left in the left eye (operated side) and normal in the right. But the oscillatory movements are distinctly and consistently different in cerebellar and in labyrinthine lesions.

	Left Half of Cerebellum	Left Labyrinth
Deviation of eyes	To right	To left
Movement of eyes	Quick oscillations in any plane, especially when disturbed or when an object is looked at	Slow movement to left with quick return to right (Labyrinthine nystagmus)
	If head is turned 90° to left oscillations are increased	If head is turned 90° to left, nystagmus is diminished
	No twitching of M. corrugator supercilii medialis	Marked twitching of M. corrugator supercilii medialis
Attitude	Left pleurosthotonus	Torsion of head so that occiput is approximated to left shoulder
	Snout to left	Snout to right
	Prefers to lie on right side	Prefers to lie on left side
Part affected	Posterior chiefly	Anterior chiefly

The Effect of Lesions of Cerebrum on Labyrinthine Reactions:—

Removal of a part or of the whole of the cerebellum made it obvious that the deviation of the eyes and the nystagmus resulting from labyrinthine stimulation or destruction was little if at all influenced directly from the cerebellum, and further that the pathway to the eye nuclei lay outside the cerebellum. It therefore became necessary to ascertain—

(1) Whether the path from the labyrinth to the eye nuclei is direct by the fasciculus longitudinalis medialis (posterior longitudinal bundle) or whether other higher centres are involved.

(2) Whether removal of various parts of the cortex of the cerebrum changes the labyrinthine effects. Since the quick component in the labyrinthine nystagmus could occur and could be produced only in the conscious state, suggesting a cerebral origin, it was determined to ascertain—

(3) From what portion of the cerebrum the quick component comes.

To accomplish this it was necessary to remove various parts of the cerebrum, and either immediately after or at a later date to observe the effects of labyrinthine stimulation or destruction.

The following as well as other experiments were done:

1. Destruction of both occipital lobes of the cerebrum.
2. Destruction of approximately half of the cerebral cortex.
3. Removal of entire cerebral cortex.
4. Complete removal of whole cerebrum with severance of corpora quadrigemina and splitting of cerebellum in the median line.
5. Effect of labyrinthine stimulation and destruction on decerebrated animal.

The results of these experiments may be thus summarized:—

Normal results from stimulation of the labyrinth, i. e. the deviation of the eyes can be obtained when the whole cerebrum and thalami are removed, leaving nothing but pons,

medulla, and their connections with the cranial nerves. The ocular movements in labyrinthine stimulation and after labyrinthine destruction are not merely the spasmodic flickering of a muscle, or an inaccurate and impotent movement as of a foot that never hits anything, such as pass for reflex responses in a dog with transected spinal cord, but full exact movements of normal magnitude.

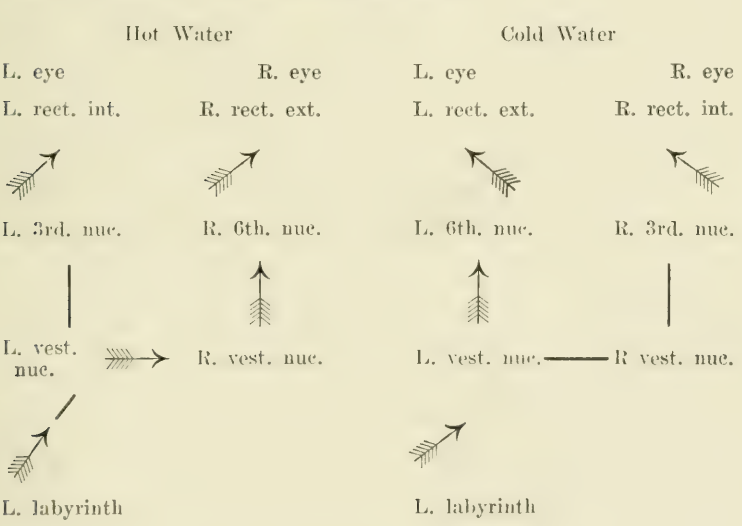
In all vertebrates so far used for experiment, and particularly in mammals, lesions of the membranous labyrinth of the ear are, without exception, attended by torsion of the anterior part of the body, and displacement of the eyes. The torsion of the head is permanent in all these forms except the human. The eye movements alone differ, and these differences are intimately associated with the increasing magnitude and complexity of development of the cerebrum in the vertebrate phylum. The typical results of lesions of the membranous labyrinth, excepting nystagmus, are very largely unaffected by the presence or absence of the phylogenetically newer pathways.

The greater capacity for adjustment following certain lesions or removals of nervous substance, inherent in the enormous development of association tracts in the central nervous system of the higher vertebrates, may tend to obscure or mask, because of the completeness of recovery following labyrinthine extirpation, some of the essential features of such a lesion in the higher forms. But the certainty and the severity of the motor disturbances immediately attending labyrinthine extirpation in the higher mammals is evidence of the persistence of the function of the vestibular mechanism. The more complete recovery in man is not necessarily to be interpreted, as Bárány believes, as evidence of the retrogression of the vestibular system in man, but may be regarded as evidence of the much greater degree of adaptability on the part of the highly complex somatic motor system, a point of view widely at variance with Bárány's.

Nystagmus occurring in the dog and cat and monkey as a result of labyrinthine stimulation or destruction, consists of

two phases—a slow deviation followed by a quick return—varying definitely in direction with the labyrinth which is stimulated or destroyed. As we have shown, these phases are distinct. Thus the slow deviation is not affected by removal of the cerebellum, nor is it affected by complete removal of the anterior corpora quadrigemina, and longitudinal splitting of cerebellum. In short, if the anatomical path from the labyrinth through the vestibular nuclei to the eye nuclei (posterior longitudinal bundle) be intact, the deviation can be produced by stimulation, and appears after destruction, so soon as the animal begins to pass out from under the influence of the narcotic. Deviation appears at a time when the reflex responses of the leg muscles are completely in abeyance, and paripassu with the corneal reflex.

The following diagrams illustrate the action of hot or cold water to produce the deviations; the experiments on which they are based will be published later:—



The quick phase of nystagmus, though not affected by the removal of the cerebellum or of the greater part of the occipital cortex, is affected by removal of the whole cerebral

cortex. It does not occur in animals completely decerebrated, including optic thalamus, even though the oculovestibular tract be unimpaired. Further, it appears later than the deviation, and can only be produced, or will only occur, when the animal emerges from the effect of the anesthetic.

We have attempted in dogs, cats and monkeys to find some region in a hemisphere where a lesion will modify or abolish the nystagmus which follows stimulation or destruction of the labyrinth. In the course of the experiments we have removed the various areas of the cortex, frontal, parietal, occipital, and temporal, and have also at times destroyed the subcortical ganglionic masses. Having destroyed these areas, we have then stimulated one or both labyrinths with the electrical current, with hot and with cold water, and by rotation. We have also in many of these cases destroyed one labyrinth either concomitantly with or subsequent to the cerebral lesion—a labyrinth either on the side of, or opposite to, the cerebral lesion. We cannot at this time enter into the details of the results, which will be published in full later, but we desire to state here that the only region where we obtain consistent alteration of the labyrinthine simulation, and that only so far as the quick component is concerned, is the region of, or adjacent to, the temporal lobe. In these cases we found that stimulation of the labyrinth on the side opposite the cerebral lesion with cold water or with the anode, gave typical labyrinthine nystagmus lateral in character, while stimulation with hot water or the cathode gave deviation, but no lateral nystagmus. We also found that destruction of the labyrinth on the side of the cerebral lesion gave no nystagmus. There was further noted in some of our cases that the eye on the side of the lesion showed a rotary movement around an anteroposterior axis, while this movement was wanting in the eye of the opposide side.

To sum up, our idea of the mechanism of nystagmus is first an agency somewhere which produces a deviation of the eyes from the primary position of equilibrium or the primary position of the line of fixation of Listing. In labyrinthine nys-

tagmus this agency lies in the labyrinth. The internal rectus muscle on the side of the slow deviation and the external rectus on the opposite side are subjected to a strain greater than usual. This strain resulting in the stimulation of the sensory or afferent endings of the eye muscles, sets up afferent impulses, which are conveyed to the cerebrum along the afferent fibres in the third, fourth, and sixth cranial nerves; (Tozer and Sherrington, Proc. Roy. Soc. 1910, p. 450). These afferent impulses in their turn set up efferent impulses in the oculomotor cells of the cerebrum, which result in a quick jerky contraction of the internal rectus on the side of the slow deviation, and of the external rectus of the opposite side, with relaxation of the antagonistic muscles. The effect is a restoration of the eyes to the primary position. The eyes do not go beyond this median position when the cerebrum is intact, since the afferent impulses to contraction of the external and internal rectus muscles concerned cease when the eyes reach this position of equilibrium.

Lesions of the cerebral hemisphere on the side of the slow deviation of the eyes abolish or reduce the quick component, since it is from the hemisphere of this side that the efferent impulses concerned in the pulling of the eyes back to the median line arise.

LANTERN SLIDES WHICH WERE SHOWN.

1. Field of operation. Fig. Phil. Trans.
   (a) Dog. Trephine opening.
   (b) Dog. Normal skull.
3. Right labyrinth destroyed.
   Dog of April 25, 1909. Photographed May, 1909. Fig. 2. Roy. Soc. Trans.
4. Dog of April 25, 1909. Before second labyrinth destroyed. Photo. March 4, 1911. Fig. 2.
5. Dog of April 25, 1909. After second labyrinth destroyed. Photo. May, 1911.
6. Dog of April 25, 1909. After second labyrinth destroyed. Photo. May, 1911. Phil. Trans. Fig. 9.
7. Dog of April 25, 1909. After second labyrinth destroyed. Photo. May, 1911.

8a. Dog of Jan. 1912.
   (a) Both labyrinths destroyed. Phil. Trans. Fig. 10 and 11.

8b. (b) Both labyrinths destroyed. Photographed Nov. 1912.

9. White rat. Left labyrinth destroyed. Phil. Trans. Fig. 3.
10. White rat. Left labyrinth destroyed. Phil. Trans. Fig. 4.
11. White rat. Both labyrinths destroyed. Phil. Trans. Fig. 12.
12. White rat. Left half of cerebellum removed.
13. White rat. Right cortex of cerebrum removed. Phil. Trans. Fig. 15.
14. Dog of April 24, 1911. Left lateral lobe and posterior part of vermis of cerebellum destroyed.
15. Dog of April 24, 1911. Same dog with left labyrinth destroyed.
16. Dog of May 4, 1911. Complete destruction of cerebellum.
17. Dog of May 4, 1911. Complete destruction of cerebellum. Phil. Trans. Fig. 13.
18. Dog of May 4, 1911.
19. Dog of May 4. After left labyrinth destroyed.
20. Dog of Nov. 3, 1911. Cerebellum and left labyrinth destroyed; showed removal.
    Killed Dec. 20. Post. mort. removal of vermis, middle lobe, and large part of interior of each lateral lobe.
21.
22a. Dog of Oct. 25, 1911. Right cerebral cortex removed.
23. Dog of Oct. 25, 1911. Left labyrinth destroyed.
24. Dog of Oct. 25, 1911. Left labyrinth destroyed.
25. Dog of Oct. 25, 1911. Left labyrinth destroyed.
26. Dog.
    A. Cerebellum removed.
    B. Cerebrum removed, May 2, 1911.
    C. Normal.
27. Dog.
    A. Cerebrum removed, June 23, 1911.
    B. Half of cerebellum removed.
28. A. Intact brain.
    B. Occipital lobes of cerebrum removed. June 12, 1911.
29. Right cerebral cortex removed. Oct. 25, 1911.

Cats. Right labyrinth removed. Phil. Trans. Fig. 6. 7, 8.

(a) Pigeon after removal of cerebellum.
(b) Pigeon after removal of right labyrinth.
(c) Frog after removal of right labyrinth.
(d) Snakes in water after labyrinthine removal.

PLATES ILLUSTRATING THE PAPER OF

J. GORDON WILSON AND F. H. PIKE

ON

THE RELATION OF THE LABYRINTH TO THE

CEREBELLUM AND THE CEREBRUM.

The numbers of the figures correspond to the numbers of the lantern slides.

Diagrams:

A. Attitude of dog after removal of right half of cerebrum.

B. Antero-posterior rotation of eyes in cat after extirpation of left labyrith followed by decerebration. The nystagmus present after the left labyrinth was removed disappeared and was replaced by deviation of the eyes, most marked in left eye; can correspond to the pupillary slits.

C. Diagram of action of hot water.

D. Diagram of action of cold water.

Compare C. and D. with figures on page 406*of article. Reference may be here made to a recent article by Dejerine (Dejerine, J., Semiologie des Affections du systeme nerveux, Paris, 1914, figure 212, page 428).

1. Field of operation. Fig. from Phil. Trans., Royal Society of London, 1912, Series B, V. 203, p. 127–160.
   (a) Dog—Trephine opening.
   (b) Dog—Normal skull.
3. Dog of April 25, 1909. Right labyrinth destroyed. Photographed May, 1909. Fig. 2, Royal Society Transactions.
4. Dog of April 25, 1909. Before second labyrinth destroyed. Photographed March 4, 1911.
5. Dog of April 25, 1909. After second labyrinth destroyed. Photographed May, 1911.

8a. Dog of Jan. 1912. Both labyrinths destroyed. Photographed in 2 positions. Phil. Trans. Fig 10 and 11.

9. White rat. Left labyrinth destroyed. Phil. Trans. Fig. 3. Photographed same day.
10. White rat. Left labyrinth destroyed. Phil. Trans. Fig. 4. Photographed same day.
11. White rat. Both labyrinths destroyed. Phil. Trans. Fig. 12. Photographed same day.
12. White rat. Left half of cerebellum removed. Photographed same day.
13. White rat. Right cortex of cerebrum removed. Phil. Trans. Fig. 15. Photographed same day.
17. Dog of May 4, 1911. Complete destruction of cerebellum. Phil. Trans. Fig. 13.

26 A. Dog of May 4, 1911. Photograph of brain showing removal of whole of cerebellum.

27. B. Dog of June 23, 1911. Photograph of brain showing removal of left half of cerebellum.

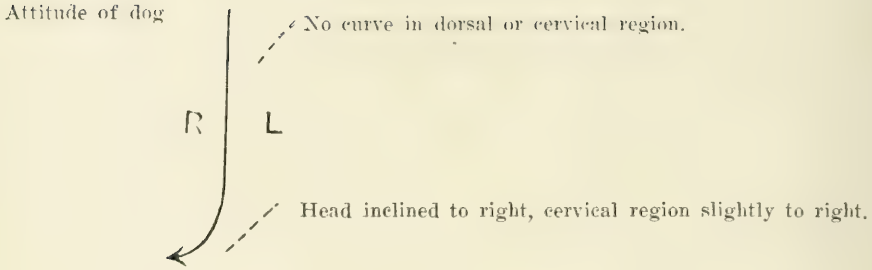
*Page 9 of this Supplement.

28. Dog. Photographs showing—
A. Normal brain.
B. Brain showing removal of both occipital lobes of cerebrum. June 12, 1911.

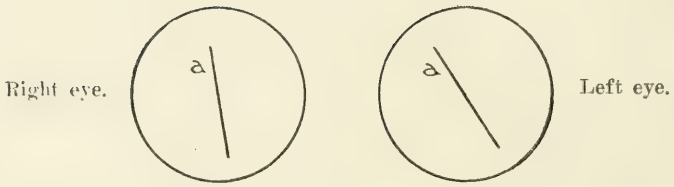
30. Cat. Right labyrinth removed. Roy. Soc. Phil. Trans. Fig. 6, 7, 8.
(a) Removal of right labyrinth. Photographed while sitting quietly on table with eyes shut. The eyes were opened at the first click of the shutter and remained opened during the exposure. Exposure, $\frac{1}{4}$ second.
(b) Removal of right labyrinth. Exposure 1/10 second.
(c) Removal of right labyrinth. At the first click of the shutter the cat turned its head to the right (the auditory reinforcement). The slight blurring of the photograph seen about the eyes, and particularly about the left eye shows the character and extent of the movement. Exposure $\frac{1}{4}$ second.

31. Dog. Nov. 4, 1913. Removal of part of left lobe of Cerebellum. Photographed Nov. 22, 1913. On Nov. 21st. it was noted: "Dog is able to walk about and head is coming back to normal. At times the eye movements are very marked, and may be either quick upwards or to right. These do not correspond to labyrinthine nystagmus, but are oscillations which vary in intensity and which appear in any plane."

32. Monkey. Extreme torsion of head after ablation of left labyrinth and left temporal lobe. Left labyrinth destroyed on Nov. 7, 1913. Marked labyrinthine nystagmus followed which had disappeared on 10th November. Left temporal lobe removed on Nov. 26, 1913, when the attitude of the head was very marked; nystagmus reappeared as animal was passing under the anaesthetic. Four hours after operation the eyes were deviated to left and down, and nystagmus was present. On Nov. 27, 1913, the animal was sitting up with the eyes at rest, but deviated to left; no nystagmus was present.


Attitude of dog
No curve in dorsal or cervical region.
R
L
Head inclined to right, cervical region slightly to right.

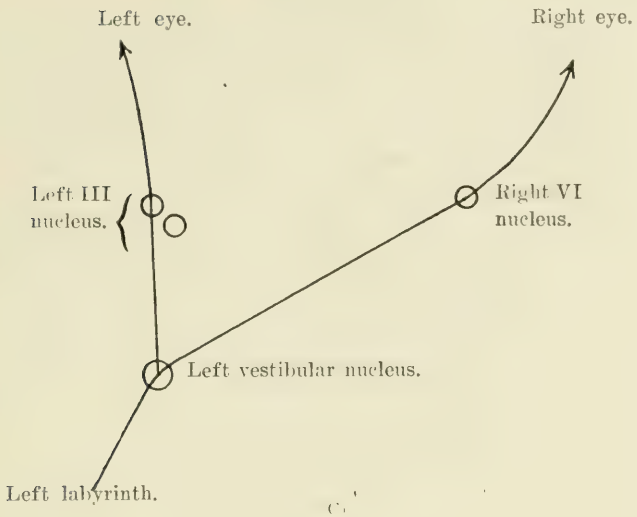
A


Right eye.
a
a
Left eye.

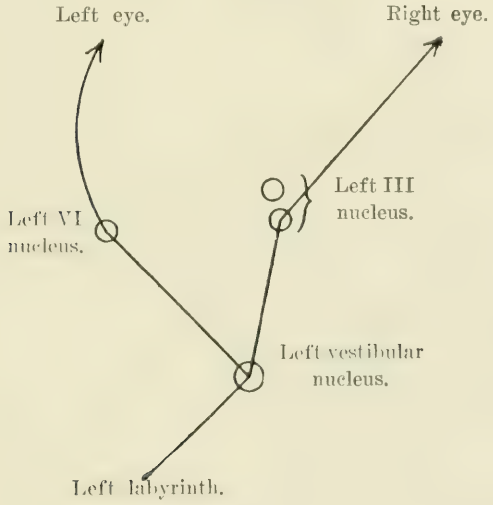
B

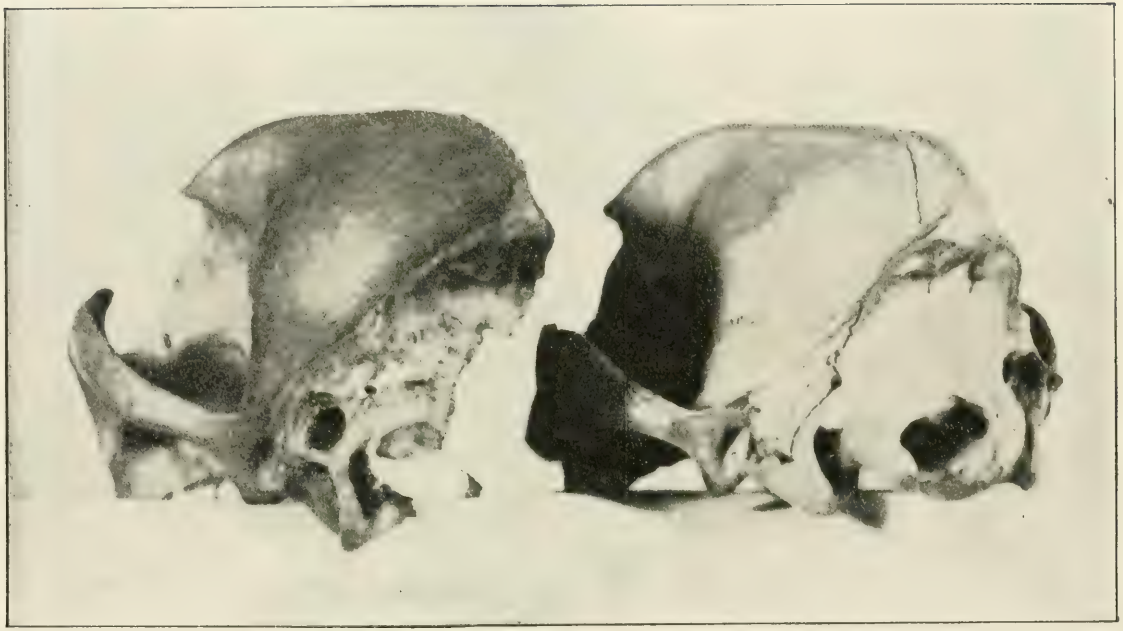
Diagram of Action of Hot and Cold Water.

Hot water.



Cold water.





(a) (b)

Fig. 1.

Fig. 1.

Fig. 3.

Fig. 8(a).

Fig. 5.

Fig. 9

Fig. 8(a).

Fig. 11

Fig. 10.

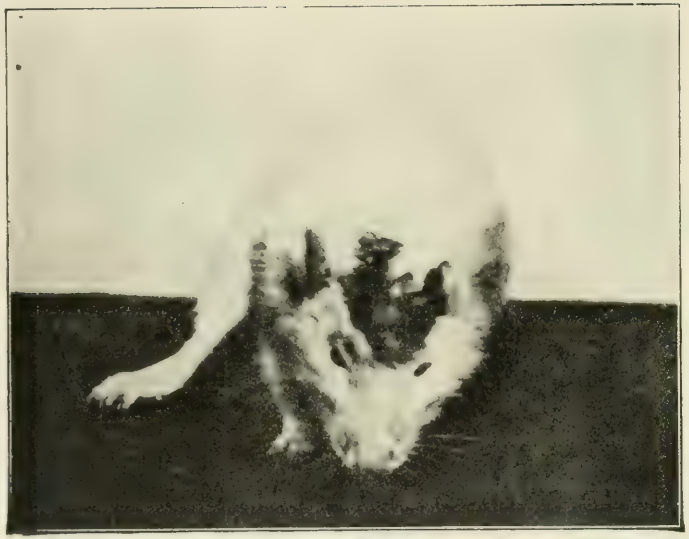


Fig. 13.

Fig. 12.

Fig. 17.

Fig. 28(a).

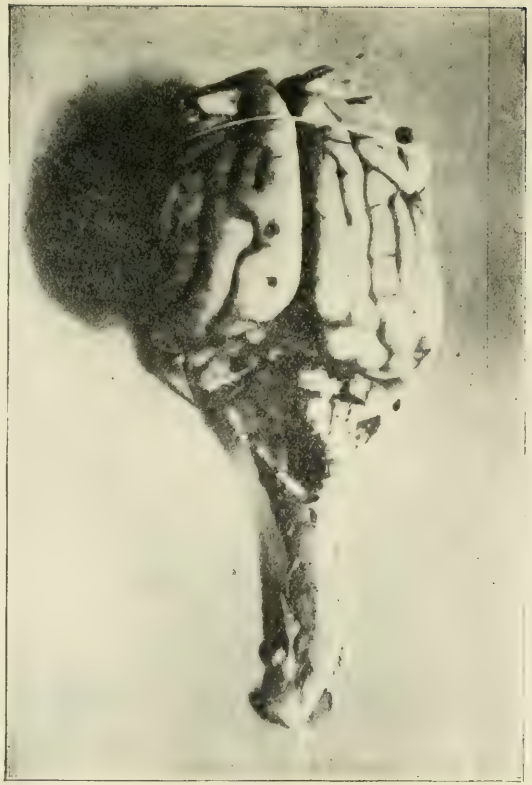


Fig. 27(b).

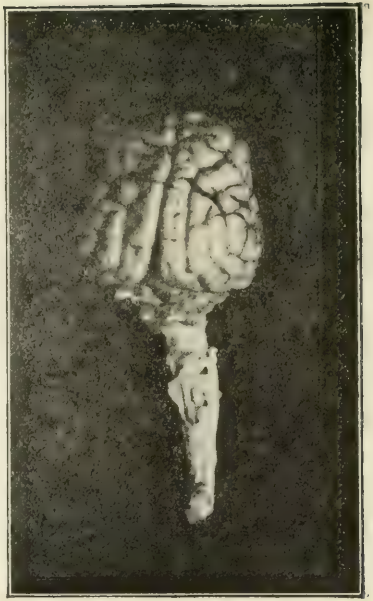


Fig. 26(a).

Fig. 28(b).

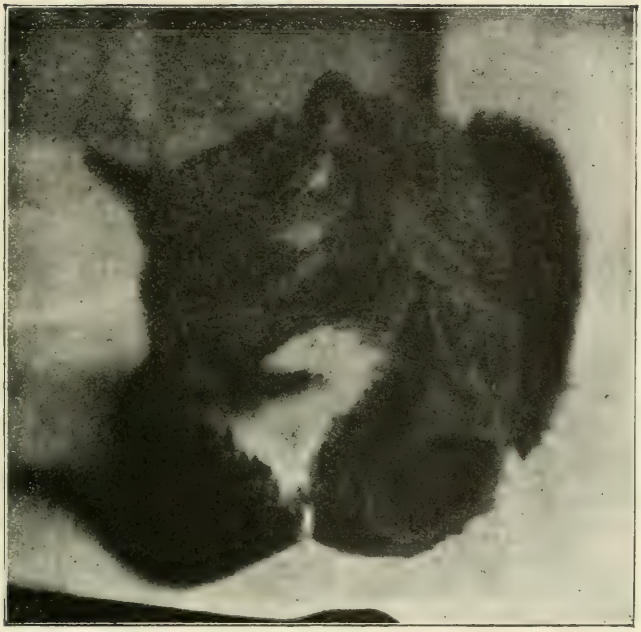


Fig. 30(a).

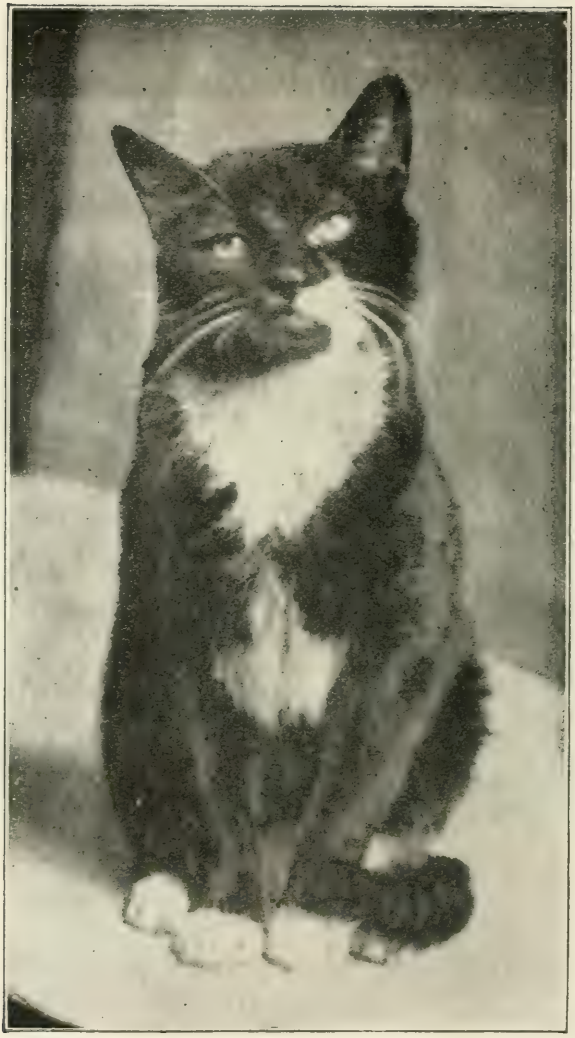


Fig. 30(b).

Fig. 30(c).

Fig. 31.

Fig. 32.

TRANSACTIONS

OF THE

AMERICAN OTOLOGICAL SOCIETY,

FORTY-EIGHTH ANNUAL MEETING.

CLIFTON HOTEL,

NIAGARA FALLS, ONT., CANADA.

JUNE 3 and 4, 1915.

VOLUME XIII. PART III.

PUBLISHED BY THE SOCIETY:
MERCURY PUBLISHING COMPANY, PRINTERS.
NEW BEDFORD, MASS.
1915.

Extra copies may be had by addressing the secretary, Dr. John B. Rae, 247 West 70th Street, New York, N. Y., or Mercury Publishing Company, 112 Union Street, New Bedford, Mass.

CONTENTS.

Members of the American Otological Society 415
Minutes of the Forty-Eighth Annual Meeting 420
Report of Two Cases of Mastoid Abscess, Infective Thrombosis of the Lateral Sinus and Jugular Vein; Resection of the Jugular; Recovery. By J. H. Bryan, M. D., Washington, D. C 424
Report of Spontaneous Cure of Six Cases of Unrecognized Sinus Thrombosis, Accidentally Discovered During Operations. By Ewing W. Day, M. D., Pittsburg, Pa. 436
Discussion 443
Nerve Deafness and Mumps. By Thomas Hubbard, M. D., Toledo, Ohio 451
Focal Infection in the Etiology of Labyrinth Disease. By George E. Shambaugh, M. D., Chicago, Ill 462
Discussion 468
The Differential Diagnosis of Lesions of the Labyrinth and Cerebellum. By J. Gordon Wilson, M. A., M. B., Chicago, Ill 470
Discussion 486
A Bloodless Method of Enucleating Tonsils. By E. Terry Smith, M. D., Hartford, Conn 489
Discussion 502
Decompression for the Relief of Choked Disc Following Thrombosis of the Lateral Sinus. By Harry Friedenwald, M. D., Baltimore, Md., and J. W. Downey, Jr., M. D., Baltimore, Md 503
The Ear-Tests of Barany in Locating Cerebellar and Other Encephalic Lesions. By B. Alexander Randall, M. D., and Isaac H. Jones, M. D., Philadelphia, Pa 511
Discussion 520
The Chorda-Tympani Nerve in Otology. By Charles E. Perkins, M. D., New York, N. Y 522
Discussion 528

An Improved Technique in the Application of the Thiersch Graft in the Radical Operation for Chronic Middle Ear Suppuration. By Edward Bradford Dench, M. D., New York, N. Y. 529

Discussion 531

Treatment of Chronic Suppurative Otitis Media, with Report of Cases. By Ernest de Wolfe Wales, Indianapolis, Indiana 534

Discussion 538

Somnoform as an Anesthetic. By T. H. Halsted, M. D., Syracuse, N. Y. 544

Discussion 551

A Modified Radical Mastoid Operation. By J. A. Stucky, M. D., Lexington, Ky. 553

Discussion 558

Acute Suppurative Otitis Media with Unusual Complications. By Eugene R. Lewis, M. D., Dubuque, Iowa 561

Discussion 565

Report of a Case of Brain Abscess with Unusual Findings. By George L. Richards, M. D., Fall River, Mass. 568

Discussion 573

Acute Hemorrhagic Mastoiditis. By Hanau W. Loeb, M. D., St. Louis, Mo. 576

Transitional Epithelium in the Rhinopharynx. By W Sohier Bryant, A. M., M. D., F. A. C. S., New York, N. Y. ... 582

Some Remarks on the X-Ray Diagnosis of Mastoiditis. By Emil Amberg, M. D., Detroit, Mich. 598

Discussion 641

MEMBERS

OF THE

AMERICAN OTOLOGICAL SOCIETY.

Names of deceased members while in membership in full face.

HONORARY MEMBERS.

Prof. A. Graham Bell.....1336 19th St.....Washington, D. C.....
Dr. Albert H. Buck.....Hewetts.....Long Island, N. Y....
Dr. E. H. Clarke.
Dr. J. Orne Green.....267 Newbury St.....Boston, Mass.....
Dr. Edw. L. Holmes.
Dr. Hermann Knapp.
Sir William Macewen.....3 Woodside Crescent.....Glasgow, Scotland....
Dr. Arthur Mathewson.....Washington, D. C....
Prof. A. F. Mayer.
Prof. Adam Politzer.....1 Gongazagesse Sq.....Vienna, Austria.....
Dr. Thomas R. Pooley.....40 East 41st St.....New York N. Y.....
Dr. Jonathan S. Prout.....Garden City, L. I....
Dr. H. Luc.....54 Rue de Varenne.....Paris, France.....
Dr. Samuel Theobald.....Cathedral & Howard Sts...Baltimore, Md.....
Dr. E. Williams.

MEMBERS.

Dr. Frank W. Abbott.
Dr. Alpheus E. Adams.....Balmville, Cor. North St...Newburg, N. Y.....
Dr. John L. Adams.
Dr. C. R. Agnew.
Dr. W. F. Akin.
Dr. Henry A. Alderton.....915 Channing Ave.....Palo Alto, Cal.....
Dr. H. P. Allen.
Dr. Alfred M. Amadon*....543 Boylston St.....Boston, Mass.....
Dr. Emil Amberg*.....David Whitney Bldg.....Detroit, Mich.....
Dr. Joseph A. Andrews.....Santa Barbara, Cal...
Dr. Stephen C. Ayres.....4 West 7th St.....Cincinnati, Ohio.....
Dr. Gorham Bacon.....47 West 54th St.....New York, N. Y.....
Dr. Robert Barclay.....3894 Washington Boulevard.St. Louis, Mo.....
Dr. John F. Barnhill.....Rms. 404-411 Pennway Bldg.Indianapolis, Ind.....
Dr. Edwin W. Bartlett.
Dr. T. Passmore Berens....35 Park Ave.....New York, N. Y.....
Dr. Herbert S. Birkett.....252 Mountain St.....Montreal, Canada.....

Dr. Hugh B. Blackwell*...148 West 58th St.....New York City.....
Dr. Clarence J. Blake.....226 Marlborough St.....Boston, Mass.....
Dr. Frank J. Blodgett.....140 West 72nd St.....New York, N. Y.....
Dr. Charles R. C. Borden*..484 Beacon St.....Boston, Mass.....
Dr. William C. Braislin....556 Washington Ave.....Brooklyn, N. Y.....
Dr. Joseph H. Bryan.....818 17th St. N. W.....Washington, D. C.....
Dr. W. Sohier Bryant.....19 West 54th St.....New York, N. Y.....
Dr. Ernest U. Buckman....99 South Franklin St.....Wilkes Barre, Pa.....
Dr. Charles H. Burnett.
Dr. Ralph Butler.....1824 Chestnut St.....Philadelphia, Pa.....
Dr. R. Bishop Canfield*.....Ann Arbor, Mich.....
Dr. Franklin P. Capron.....118 Angell St.....Providence, R. I.....
Dr. James B. Clemens.....10 East 71st St.....New York, N. Y.....
Dr. Cornelius G. Coakley...53 West 56th St.....New York, N. Y.....
Dr. William Cowen.....35 East 60th St.....New York, N. Y.....
Dr. Charles N. Cox.....257 Jefferson Ave.....Brooklyn, N. Y.....
Dr. Eugene A. Crockett.....298 Marlborough St.....Boston, Mass.....
Dr. J. Frank Crouch.....513 North Charles St.....Baltimore, Md.....
Dr. John P. Davidson.
Dr. Ewing W. Day.....1005 Westinghouse Bldg....Pittsburg, Pa.....
Dr. Edward B. Dench.....15 East 53rd St.....New York, N. Y.....
Dr. J. H. Dix.
Dr. Ernest E. Doble.....52 Cross St.....Quincy, Mass.....
Dr. Arthur B. Duel.....27 East 57th St.....New York, N. Y.....
Dr. John Dunn*.....314 East Franklin St.....Richmond, Va.....
Dr. E. Dwyer.
Dr. Wells P. Eagleton.....15 Lombardy St.....Newark, N. J.....
Dr. E. P. Ely.
Dr. Joseph B. Emerson.....40 East 41st St.....New York, N. Y.....
Dr. George F. Fiske.....25 East Washington St....Chicago, Ill.....
Dr. J. R. Fletcher*.....25 East Washington St....Chicago, Ill.....
Dr. Edw. Fridenberg.
Dr. Percy Fridenberg.....38 West 59th St.....New York, N. Y.....
Dr. Harry Friedenwald....1029 Madison Ave.....Baltimore, Md.....
Dr. Blancome E. Fryer.
Dr. Max A. Goldstein.....3858 Westminster Place...St. Louis, Mo.....
Dr. John Green.
Dr. Emil Gruening.
Dr. George W. Hale.....Wilcox Building.....Nashville, Tenn.....
Dr. Thomas H. Halsted.....831 University Block.....Syracuse, N. Y.....
Dr. Philip Hammond.....483 Beacon St.....Boston, Mass.....
Dr. Herbert Harlan.....516 Cathedral St.....Baltimore, Md.....
Dr. Thomas J. Harris.....104 East 40th St.....New York, N. Y.....
Dr. David Harrower.....9 Elm St.....Worcester, Mass.....
Dr. N. Darrell Harvey.....114 Watermann St.....Providence, R. I.....

Dr. William H. Haskin.....40 East 41st St.....New York, N. Y.....
Dr. R. Johnson Held*.....616 Madison Ave.....New York, N. Y.....
Dr. Neil J. Hepburn.....317 West 23rd St.....New York, N. Y.....
Dr. James Hewitt.
Dr. Christian R. Holmes....8 East 8th St.....Cincinnati, Ohio.....
Dr. Edgar M. Holmes*.....531 Beacon St.....Boston, Mass.....
Dr. Erastus E. Holt.....723 Congress St.....Portland, Me.....
Dr. Frank T. Hopkins*....182 West 58th St.....New York City.....
Dr. Thomas Hubbard.....515 Nicholas Bldg.....Toledo, O.....
Dr. J. M. Ingersoll*.....1021 Prospect Ave.....Cleveland, Ohio.....
Dr. Frederick L. Jack.....215 Beacon St.....Boston, Mass.....
Dr. Walter B. Johnson.....170 Broadway.....Paterson, N. J.....
Dr. Richard Jordan.....165 West 58th St.....New York, N. Y.....
Dr. Joseph A. Kenefick.....78 East 54th St.....New York, N. Y.....
Dr. B. R. Kennon.....64 Granby St.....Norfolk, Va.....
Dr. Philip D. Kerrison.....58 West 56th St.....New York, N. Y.....
Dr. Gordon King.
Dr. Charles J. Kipp.
Dr. D. Braden Kyle.....1517 Walnut St.....Philadelphia, Pa.....
Dr. J. W. Lawton.
Dr. George A. Leland.....354 Commonwealth Ave....Boston, Mass.....
Dr. John C. Lester.....432 Madison Ave.....New York, N. Y.....
Dr. Eugene R. Lewis*.....1130 Main St.....Dubuque, Iowa .....
Dr. Robert Lewis .....48 West 40th St.....New York, N. Y.....
Dr. W. S. Little.
Dr. Hanau W. Loeb.....537 N. Grand Ave.....St. Louis, Mo.....
Dr. James E. Logan.....1208 Wyandotte St.....Kansas City, Mo.....
Dr. Francis B. Loring.....1420 K St., N. W.....Washington, D. C.....
Dr. David B. Lovell.....Rooms 407-408 State Mutual Building .....Worcester, Mass.....
Dr. C. J. Lundy.
Dr. Stephen H. Lutz*.....284 Hancock St.....Brooklyn, N. Y.....
Dr. G. Hudson-Makuen....1627 Walnut St.....Philadelphia, Pa....
Dr. George B. McAuliffe....616 Madison Ave.....New York, N. Y.....
Dr. James F. McCaw.....Bank & Loan Bldg.....Watertown, N. Y.....
Dr. Read J. McKay.....9th & Market Sts.....Wilmington, Del.....
Dr. James F. McKernon....62 West 52nd St.....New York, N. Y.....
Dr. O. A. M. McKimmie....1330 Mass. Ave. N. W.....Washington, D. C....
Dr. Cyrus S. Merrill.....27 South Hawk St.....Albany, N. Y.....
Dr. Horace J. Miller.
Dr. W. W. Moreland.
Dr. Henry L. Morse.....112 Marlborough St.....Boston, Mass.....
Dr. Harris P. Mosher.....828 Beacon St.....Boston, Mass.....
Dr. Willis B. Moulton*.....180 State St.....Portland, Me.....
Dr. Robert C. Myles.....46 West 38th St.....New York, N. Y.....
Dr. James E. H. Nichols.

Dr. Henry D. Noyes.

Dr. J. F. Noyes.

Dr. Francis R. Packard....304 South 19th St.....Philadelphia, Pa.....
Dr. John R. Page*.....127 East 62d St.....New York, N. Y.....
Dr. Charles E. Perkins*....125 West 82nd St.....New York, N. Y.....
Dr. Wendell C. Phillips.....40 West 47th St.....New York, N. Y.....
Dr. Norval H. Pierce.....22 E. Washington St.....Chicago, Ill.....
Dr. Edward M. Plummer....5 Adams St.....Charlestown, Mass....

Dr. Oren D. Pomeroy.

Dr. Barton H. Potts.

Dr. George H. Powers, Jr.*.298 Marlborough St.....Boston, Mass.....
Dr. John B. Rae.....247 West 70th St.....New York, N. Y.....
Dr. B. Alexander Randall...1717 Locust St.....Philadelphia, Pa.....

Dr. Wm. Rankin, Jr.

Dr. J. M. Ray.....Atherton Building.....Louisville, Ky.....
Dr. Henry O. Reik.....506 Cathedral St.....Baltimore, Md.....
Dr. W. Scott Renner.....341 Linwood Ave.....Buffalo, N. Y.....
Dr. George L. Richards.....124 Franklin St.....Fall River Mass.....
Dr. Huntington Richards..St. Paul's School.....Concord, N. H.....
Dr. John D. Richards*.....44 West 49th St.....New York, N. Y.....
Dr. Charles W. Richardson..1317 Connecticut Ave.....Washington, D. C.....
Dr. Stephen O. Richey.....732 17th St.....Washington, D. C.....

Dr. Charles E. Rider.

Dr. F. W. Ring.

Dr. Henry W. Ring.....185 Church St.....New Haven, Conn....
Dr. Edmund C. Rivers.....1632 Welton St.....Denver, Col.....
Dr. Arthur W. de Roaldes...624 Gravier St.....New Orleans, La.....
Dr. Walter Roberts.....1732 Spruce St.....Philadelphia, Pa.....
Dr. William K. Rogers....88 East State St.....Columbus, Ohio.....

Dr. D. B. St. John Roosa.

Dr. John D. Rushmore.....129 Montague St.....Brooklyn, N. Y.....
Dr. W. E. Sauer*.....201 Humboldt Bldg.....St. Louis, Mo.....
Dr. T. Laurance Saunders*.45 West 50th St.....New York City.....

Dr. Wm. W. Seely.

Dr. Ralph W. Seiss.....255 South 17th St.....Philadelphia, Pa.....

Dr. S. Sexton.

Dr. George E. Shambaugh..People's Gas Bldg.....Chicago, Ill.....
Dr. John B. Shapleigh.....Grand & Washington Aves..St. Louis, Mo.....
Dr. Warren S. Shattuck....160 Clinton St.....Brooklyn, N. Y.....

Dr. Henry L. Shaw.

Dr. John E. Sheppard.....130 Montague St.....Brooklyn, N. Y.....
Dr. Burt R. Shurly*.....32 Adams Ave. W.....Detroit, Mich.....
Dr. E. Terry Smith.....36 Pearl St.....Hartford, Conn.....
Dr. S. MacCuen Smith.....1429 Spruce St.....Philadelphia, Pa.....
Dr. Sargent F. Snow.....707 University Block.....Syracuse, N. Y.....

Dr. Edmund D. Spear.....100 Boylston St.....Boston, Mass.....
Dr. Horatio N. Spencer.....2725 Washington Ave.....St. Louis, Mo.....
Dr. Selden Spencer*.....2725 Washington Ave.....St. Louis, Mo.....
Dr. Frank Sprague.
Dr. Franklin M. Stephens...19 West 54th St.....New York, N. Y.....
Dr. George T. Stevens.....350 West 88th St.....New York, N. Y.....
Dr. Samuel B. St. John.
Dr. George Strawbridge.
Dr. Joseph A. Stucky.....Lexington, Ky.....
Dr. Edward Blair Sutphen*.162 South St.....Morristown, N. J.....
Dr. Theron Y. Sutphen....999 Broad St.....Newark, N. J.....
Dr. Lewis H. Taylor.....82 South Franklin St.....Wilkes Barre, Pa.....
Dr. J. J. Thomson*.....40 West 47th St.....New York City.....
Dr. William Thomson.
Dr. Robert Tilley.
Dr. Andrew Timberman*...112 East Broad St.....Columbus, Ohio.....
Dr. George L. Tobey*.....416 Marlborough St.....Boston, Mass.....
Dr. J. J. B. Vermyne.
Dr. Ernest de Wolfe Wales..605 Hume-Mansur Bldg....Indianapolis, Ind.....
Dr. D. Harold Walker.....390 Commonwealth Ave....Boston, Mass.....
Dr. David Webster.....24 East 48th St.....New York, N. Y.....
Dr. Joseph A. White.....200 East Franklin St.....Richmond, Va.....
Dr. Edward Wheelock.....26 Gibbs St.....Rochester, N. Y.....
Dr. Fred Whiting.....19 West 47th St.....New York, N. Y.....
Dr. Frederick M. Wilson...834 Myrtle Ave.....Bridgeport, Conn....
Dr. John Gordon Wilson....104 S. Michigan Ave.....Chicago, Ill.....
Dr. Hiram Woods.....842 Park Ave.....Baltimore, Md.....

*Associate.

American Otological Society.

The forty-eighth annual meeting of the American Otological Society was held at the Hotel Clifton, Niagara Falls, Canada. June 3rd and 4th, 1915.

The following members and guests registered during the meeting:

Members.

CHAS. W. RICHARDSON
J. H. BRYAN
J. B. SHAPLEIGH
GEO. F. FISKE
EDWARD B. DENCH
O. A. M. McKIMMIE
SARGENT F. SNOW
JOHN F. BARNHILL
G. A. LELAND
B. ALEX. RANDALL
HANAU W. LOEB
W. P. EAGLETON
JAMES E. LOGAN
JOHN G. WILSON
WM. C. BRAISLIN
H. FRIEDENWALD
THOMAS HUBBARD
ROBERT LEWIS
GEO. L. RICHARDS
E. U. BUCKMAN
M. A. GOLDSTEIN
T. H. HALSTED
E. TERRY SMITH
G. HUDSON-MAKUEN
W. SCOTT RENNER
W. S. SHATTUCK
E. DE WOLFE WALES
H. P. MOSHER
JOHN B. RAE
FREDERICK L. JACK
RALPH BUTLER
GEO. SHAMBAUGH
JAS. F. McKERNON
J. A. STUCKY
EWING W. DAY

Associate Members.

EMIL AMBERG
FRANK T. HOPKINS
CHAS. E. PERKINS
GEORGE H. POWERS, JR.
EUGENE R. LEWIS

Guests.

R. C. LYNCH
HENRY L. SWAIN
OTTO FREER
JOHN F. FAIRBAIRN

Thursday Afternoon, June 3rd, 1915.

Executive Session.

The meeting was opened by Dr. Charles W. Richardson, President, when the minutes of the forty-seventh annual meeting were read and approved.

The President appointed as the Business Committee, Drs. E. B. Dench, Ewing W. Day and Thomas H. Halsted.

The report of the Secretary was read and approved, as was also the report of the Treasurer.

The President appointed as a Committee of Necrology, Drs. Robert Lewis and Gorham Bacon.

On motion, the thanks of the Society were extended to Dr. W. S. Renner and to Dr. A. Sharp for their valuable assistance in obtaining the use of the motion-picture machine.

Scientific Session.

Papers were read by Drs. Jos. H. Bryan, Ewing W. Day, Thomas Hubbard, George E. Shambaugh, J. Gordon Wilson, E. T. Smith, B. Alexander Randall.

Friday, June 4th, 9 A. M.

Scientific Session.

Papers were read by Drs. Chas. E. Perkins, Edward B Dench, E. de Wolfe Wales, Thos. H. Halsted, E. R. Lewis and J. A. Stucky.

Friday, June 4th, 2 P. M.

Executive Session.

The Business Committee reported the nomination of officers for 1915–1916:

For President—Dr. Chas. W. Richardson, Washington, D. C.

For Vice-President—Dr. C. R. Holmes, Cincinnati, O.

For Secretary-Treasurer—Dr. John B. Rae, New York, N. Y.

For Committee on Membership—Dr. Frederick L. Jack, to serve the unexpired term of Dr. H. S. Birkett; Dr. R. Lewis, to serve two years; Dr. James F. McKernon, to serve three years.

For Committee on Publication—Dr. B. Alex. Randall, Dr. Arthur B. Duel, the Secretary, *ex-officio*.

On motion, these officers were duly elected.

The Business Committee further recommended to the Society that the President and Vice-President should not hold office for more than one year consecutively. On motion, this was duly carried.

The President appointed as the Standing Committee, Dr. W. P. Eagleton and Dr. F. R. Packard.

The Committee on Membership reported favorably upon the nominations of Dr. J. Ingersoll, Cleveland, O.; Dr. W. E. Sauer, St. Louis, Mo.; and Dr. J. R. Fletcher, Chicago, Ill.

On motion, these gentlemen were duly elected to membership in the Society.

The Committee on Membership further recommended that the applications of Dr. A. F. Koetter, St. Louis, and Dr. Benj. D. Parish, Philadelphia, be held over for one year.

On motion, this recommendation was sustained by the Society.

The following nominations for membership were made:

Dr. Francis B. Emerson of Boston, Mass. Proposed by Dr. Harris P. Mosher; seconded by Dr. George L. Richards.

Dr. L. W. Dean of Iowa City, Iowa. Proposed by Dr. John Gordon Wilson; seconded by Dr. Geo. E. Shambaugh.

Dr. I. A. Jones of Philadelphia, Pa. Proposed by Dr. Geo. E. Shambaugh; seconded by Dr. J. Gordon Wilson.

Dr. Duncan MacPherson of New York, N. Y. Proposed by Dr. John B. Rae; seconded by Dr. James F. McKernon.

Dr. William L. Culbert, New York, N. Y. Proposed by Dr. James F. McKernon; seconded by Dr. John B. Rae.

The following excuses for absence were presented by the Secretary: Drs. Jos. A. Andrews, J. J. Thomson, G. L. Tobey, A. W. de Roaldes, J. M. Ray, J. D. Rushmore, F. R. Packard, H. O. Reik, T. L. Saunders, L. H. Taylor and W. K. Rogers.

On motion, the excuses were accepted

The Secretary presented the resignations of Dr. Edward Stieren and Dr. Richard A. Reeve.

On motion, these resignations were accepted.

Scientific Session.

Papers were read by Dr. Wm. Sohier Bryant and Dr. Emil Amberg.

In closing, the President extended to the members of the Society his appreciation of the honor done him by his election to the presidency, and his thanks for their support.

The Society then adjourned.

REPORT OF TWO CASES OF MASTOID ABSCESS, INFECTIVE THROMBOSIS OF THE LATERAL SINUS AND JUGULAR VEIN; RESECTION OF THE JUGULAR; RECOVERY.

By J. H. BRYAN, M. D., Washington, D. C.

While the first case was rather typical in character, it nevertheless presents some unusual features which justify its being placed on record. Case two, on the other hand, presents many difficulties, and is brought before the Society with the hope that the discussion will bring out some suggestions as to the best manner to manage those cases where the infected clot remains in the bulb, as in this case, where one time it seemed almost impossible to dislodge, without the radical destruction of the bone through which the lateral sinus passes into the neck. I believe it was the presence of this clot which was the source of the continued systematic infection.

Case I. Acute suppurating mastoiditis, perisinus abscess, infective thrombosis of the lateral sinus and jugular vein; resection of the jugular; streptococcus infection.

I saw this case, a girl 13 years of age, on the evening of January 25, 1914. She had been ill for two days with a marked attack of influenza. The ear became almost immediately involved, accompanied by intense pain, and the membrane ruptured spontaneously during the course of the first twenty-four hours. When I first saw her she looked ill, the temperature was 104, the left ear was discharging profusely, pus mixed with blood. She was complaining of intense pain in the ear, and there was marked tenderness over the mastoid tip.

Examination of the ear showed an intensely inflamed membrane with a perforation in the lower posterior quadrant through which a bloody purulent secretion was flowing freely. Hot boracic acid irrigations were ordered hourly during the night.

January 25. The patient was removed to the Episcopal Eye, Ear and Throat Hospital at 10 o'clock this morning. On admission her temperature was 103.4; at noon 104.4; dropping slowly to 102.8 at 8 p. m. As the child was attending a boarding school in Washington, and her parents were living some distance from the city, there was some delay in reaching them to obtain their permission for an operation.

January 26. Today the temperature ranged from 102 to 103. While the pain in the ear was greatly relieved by the hot douches, she was extremely restless and dull.

January 27. At 8 a. m., temperature 99.8. At 10 a. m., she had a severe chill, following which her temperature advanced to 105. Permission for an operation having been obtained, a simple mastoid operation was done at 2 p. m. After opening the mastoid it was found to be already very badly affected, and the antrum filled with pus. The whole of the outer wall was removed down to and including the tip of the process, and everything in the way of diseased bone was thoroughly removed with the curette. The sinus wall was exposed, carefully examined, and found to be in good condition, and as there was no reason to suspect sinus infection, it was not opened. The wound was dressed in the usual manner, and the patient returned to her room. The temperature after the operation, at 4 and at 8 p. m. was 102.

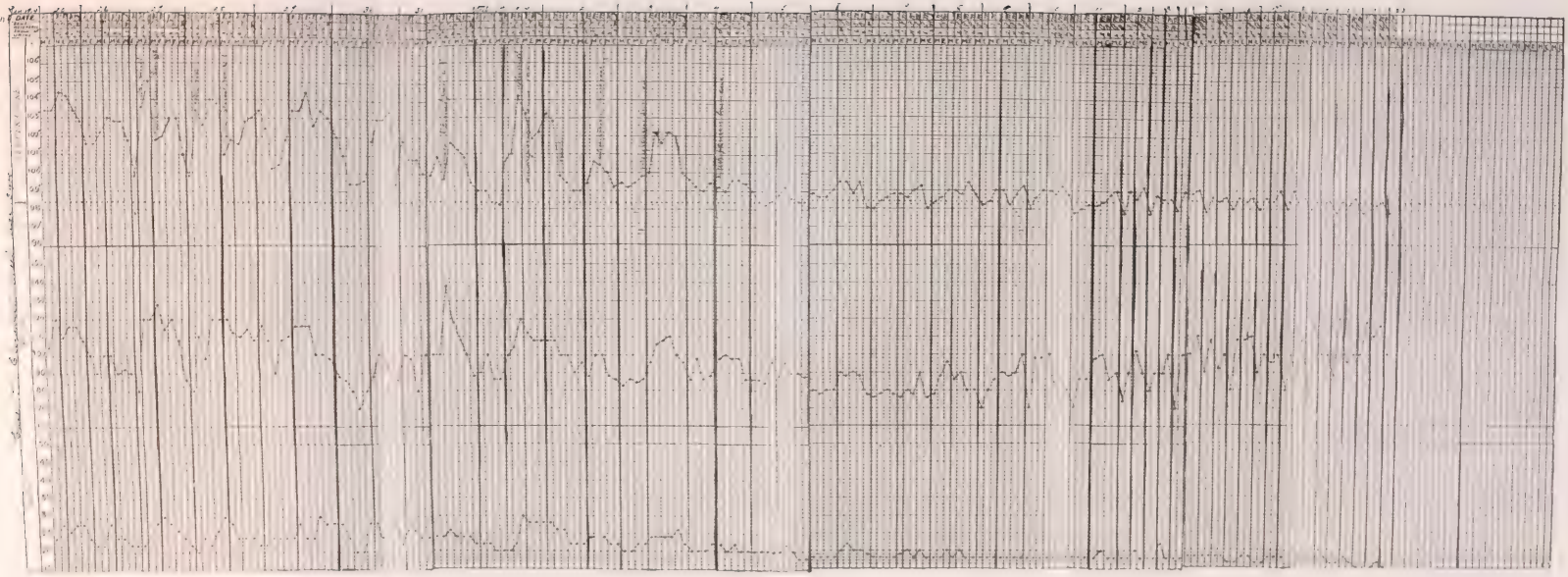
January 28. The patient this morning had a very septic appearance. Temperature at 8 a. m., 103; at 12 noon, 99.4. She had a severe chill about this time, and the temperture rose to 103.2 at 4 p. m.

January 29. At 8 a. m, temperature 104.2; at 12 noon, 104.2. Owing to the continuous high temperature, it was decided to explore the wound farther. In consultation with Dr. C. W. Richardson, who assisted me in the operation, the dressings were removed and the mastoid wound was found to be in a healthy condition. The wall of the lateral sinus was completely removed from the knee to the bulb. At the latter point, a large perisinus abscess was located. After thoroughly removing the pus and some granulations, the sinus itself was opened to its full extent from the knee to the

bulb and found filled with a firm clot. This was removed with the curette as far as the knee, and well back toward the torcular, when a sharp flow of blood followed which was controlled by compression. The curette was then passed down toward the bulb, and as there was no return flow of blood from this end of the vein we decided to remove the jugular. After packing the upper end of the lateral sinus with an iodoform gauze plug, an incision was made along the anterior border of the sterno-mastoid muscle, extending from just below the mastoid tip to near the clavicle. The vein, exposed, was found to present a firm, cord-like feeling. After ligating the vein, at its upper and lower ends, its collateral branches were then ligated, and about 2½ inches of the jugular were removed. A gauze drain was then placed in the upper and lower ends of the wound, which was closed by means of interrupted sutures. The mastoid dressings were then applied and both wounds enveloped in a bandage. The patient reacted well after operation. The temperature at 4 p. m. was 101.4. The pathologist reported he found streptococci in the clots removed from the lateral sinus and the jugular vein.

January 31. The patient since the operation has been doing well, although the temperature is markedly septic in character, and ranges from 99.2 to 104.2. The mastoid and neck wounds were dressed today and found in good condition. The gauze compress at the upper end of the lateral sinus was not removed, but the gauze wicks in the upper and lower ends of the neck wound were removed, and some pus was found in the upper end of the wound which was removed by gentle irrigation. The gauze drains were re-inserted in the neck wound and the mastoid dressings re-applied.

February 1. The patient seemed much better during the early part of the day, but at 4 p. m. she had a severe chill followed by a rapid rise of temperature to 104.2. She was given 10 c.c. of antistreptococcus serum in the lumbar region. Following the injection, the temperature became lower, ranging from 98 to 100.4 during the two following days, but owing to the continued high leucocyte count, she was given a



second injection of 10 c.c. of antistreptococcus serum on the afternoon of February 2.

February 3. Wounds redressed. Mastoid wound looking healthy. Gauze compress removed from the upper end of the lateral sinus, and as no blood followed its removal, it was not renewed. Pus found in the upper and lower ends of the neck wound after the removal of the drains. Both ends irrigated with a saturated solution of boric acid. Firm union of the edges of the wound having taken place, the stitches were removed, the gauze drains reinserted, and the mastoid dressings reapplied. The temperature during the night of the 3rd and the forenoon of the 4th ranged from 102 to 102.2. On the afternoon of the 4th, she was given 20 c.c. of the serum, and following this last injection her temperature dropped to 99.2. From this time on, the temperature became normal and remained so during the rest of her convalescence. The wounds, both the mastoid and that in her neck progressed favorably, and she was soon placed on the convalescent list. The scar in the neck for a long time remained quite insignificant, but about two months after leaving the hospital it presented quite a broad, ribbon-like appearance, and was very red. I am glad to say, however, that within the last six months this has disappeared, and the scar is insignificant. The following is a tabulated statement of the blood counts and bacteriological examination:

January 25. On admission to the hospital, leucocytes 14,400; aural pus smears showed the presence of many cocci, mostly in pairs like pneumococci, and a few in chains, like streptococci.

January 26. Leucocytes 11,600. Culture made from aural pus yesterday yields growth of streptococci.

January 28. Leucocytes, 17,300.

January 30. Leucocytes, 12,100.

January 31. Leucocytes, 15,800.

February 2. Leucocytes, 16,100.

February 4. Leucocytes, 14,100.

February 6. Leucocytes, 8,200.

Differential Count (200 counted).

Small lymphocytes, 14%.

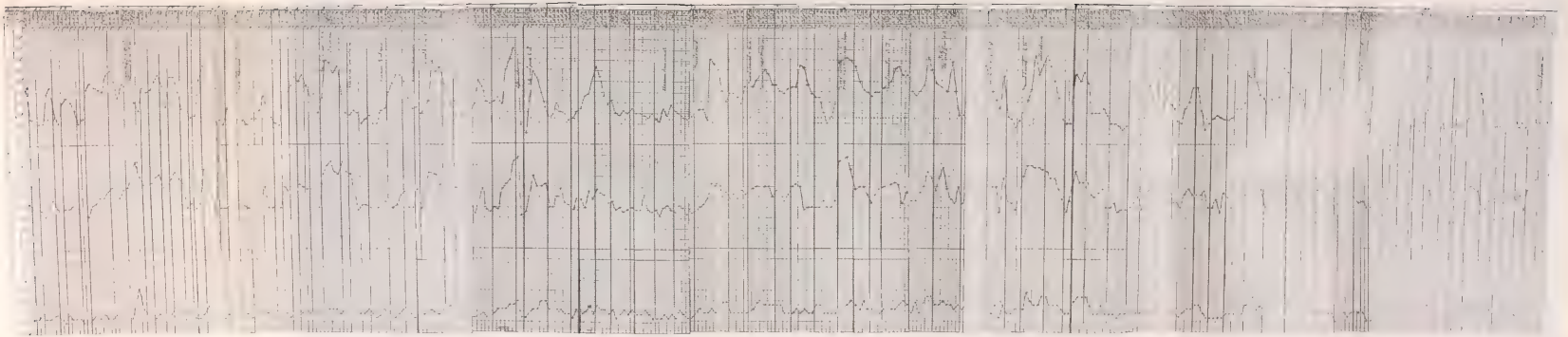
Large lymphocytes, 3%.

Polymorphoneuclears, $\frac{83}{100}$

This case presents some interesting features, among which may be mentioned the desirability or not in exposing the lateral sinus in primary mastoid operations. In the primary operation of this case, the mastoid was thoroughly cleansed out, and the bony wall covering the sinus seemed to be in a healthy state, so it was not deemed advisable to remove it. These cases, however, are very deceptive, and notwithstanding the healthy appearance of the sinus wall, there may be a nidus of diseased bone which escapes detection and from which an infection of the sinus may take place. The infection in this case took place from the perisinus abscess near the bulb, which was not detected at the time of the first operation.

Case II. Acute suppurating mastoiditis, infective thrombosis of the lateral sinus and jugular vein; resection of the jugular, metastatic abscess in the leg; staphylococcus pyogenes infection.

I saw this child, Harriet H., aged 7 years, on the evening of December 22, 1914, in consultation with her family physician, who stated that she had been ill with influenza for several days, and two days ago she complained of intense pain in the right ear. A paracentesis was done by another aurist at this time. When I saw the patient at 11:30 p. m. her temperature was 101.2, very restless, and complaining of intense pain in the ear. An examination of the ear showed the canal filled with a thick, purulent secretion, which, when wiped away, enabled me to see the drum, which was intensely swollen and red. The perforation was in the posterior quadrant, and seemed sufficiently large to admit of free drainage. There was no sagging of the posterior wall of the canal, but there was some tenderness over the mastoid. How much of this was real and how much was due to the general irritability of the child, I was



unable to state. The ear was ordered douched hourly with a hot saturated solution of boracic acid. The pain was soon relieved, and she slept through the night, having to be awakened at the stated intervals for the douching.

December 23. Today, she seemed quite comfortable, not complaining of pain except when the mastoid was pressed on firmly. The temperature at 8 a. m. was 98.4; 12 noon and 4 and 8 p. m., 98. At 11 p. m. it suddenly rose to 101.4. The ear appeared to be draining well.

December 24. The general conditions were about the same. The low morning temperature was 98, and the highest in the evening, 101.8.

December 25. At 4 a. m., she was very restless and the thermometer showed her temperature had risen to 103. At 8 a. m. it was 102; at 12, noon, 100.8. There was a decided change for the worse in the appearance of the child, and a decided tenderness over the mastoid. She was immediately taken to the Episcopal Eye, Ear and Throat Hospital, and at 2:30 p. m. a simple mastoid operation was done. The mastoid was found already badly affected, pus in the antrum, and some soft diseased bone extending through the process down to the tip; pus in considerable quantity was evacuated between the membranous and bony wall of the auditory canal where the anterior flap was well pushed forward. The mastoid process was completely exenterated down to and including the tip. The bony wall covering the lateral sinus was carefully examined, and a portion of the wall, about an eighth of an inch, was removed at about the middle of the sinus, exposing the vein, which had a healthy appearance to the eye and also on palpation. At this time, it did not appear that the sinus was involved, so the usual dressings were applied, and the patient returned to her room in good condition. Evening temperature, at 6:45, 100; 12 midnight, 99.2.

December 26. Temperature; 9 a. m., 101.4; 12 noon, 101; 4 p. m., 101.2; 8 p. m., 102.

December 28. The temperature continued to rise and fall during the past two days, showing a decided septic character.

Otherwise, the patient seemed to be doing as well as she could at this stage.

December 29. Temperature; 1 a. m., 103.6; 8 a. m., 99.4; 12 noon, 98.2; 4 p. m., 98; 8 p. m., 98.

December 30. The temperature remained low today, ranging from 98 to 98.8. The wound was dressed at 1.30 p. m. Little or no secretion found on the dressings, and the wound itself presented a very dry and glazed appearance.

January 1. The temperature continued to pursue an irregular course; 8 a. m., 100.8; 12 noon, 99; 4 p. m., 100.4. Wound dressed again, and presented a healthier appearance in that the tissue did not present such a dry appearance. Dressings somewhat moist. Temperature, 8 p. m., 104.

January 5. Temperature; 8 a. m., 100.8; 12 noon, 99; 4 p. m., 99.2; 8 p. m., 98.

January 6. The general condition of the patient remained about the same. Temperature, 8 a. m., 100.4; 12 noon, 99.6; 4 p. m., 100.2; 8 p. m., 104. The wound was dressed again today and its general appearance was satisfactory; granulations forming, and healthy in character.

January 8. Temperature; 8 a. m., 100; 12 noon, 99.8; 4 p. m., 100.4; 8 p. m., 103; and 11 p. m. it had risen to 105.

January 9. The patient passed a very restless night, and the earliest recorded temperature this morning, owing to the child being asleep, was 97.6 at 11 a. m.; 97 at 12 noon. She complained several times during the night of being chilly, following which the extremities and face were quite moist. Dr. William B. Mason saw the case in consultation, and assisted me in the operation at 1.30 p. m. Under ether anesthesia, the mastoid wound was thoroughly explored and all granulation tissue removed with the curette. The bone covering the lateral sinus was removed, exposing the sinus for some distance over the transverse sinus posteriorly and down to the bulb. At the junction of the lateral and sigmoid sinus, the vein showed a dark grayish appearance, extending back toward the torcular; from the point of exposure at the time of the original operation to the bulb, the vein presented a different appearance, being quite blue and soft to the touch.

The vein was then opened and found thrombosed, and it was then freely opened from an inch behind the sigmoid bend to the bulb, and a firm clot was removed with the curette. As no return flow of blood occurred, the curette was passed well back toward the torcular, and fragments of clot removed until a free flow of blood was obtained. The transverse sinus was then blocked off with an iodoform gauze tampon. The lateral sinus was then gently curetted as far as the bulb, until all the clot that could be reached was removed. As there was no flow of blood, an incision was made along the anterior border of the sterno-cleido mastoid muscle, extending from just below the mastoid process to within an inch of the clavicle, and the jugular vein was exposed. After separating it from its sheath, two ligatures were passed around it and tied. The thyroid and facial veins were ligated, and about 2½ inches of the jugular were removed. The wound in the neck was not closed, but packed with iodoform gauze, and a small gauze wick was passed into the sinus end of the bulb as far as possible, and the usual mastoid dressing applied. The child's condition at the time of and during the operation was very critical, the pulse being 148 and the respiration 30. Although she lost very little blood, she was very weak. Dr. Blair Spencer, her family physician, gave her strychnine sulphate, grains 1/60, hypodermically, and administered 700 c.c. normal salt solution through the median cephalic vein, after which she rallied and was in fair condition when returned to her room. The operation lasted a little over one hour. The jugular vein, when opened, was found to be filled to its full extent with a solid clot. A culture made from the clots taken from the lateral sinus, the jugular vein and from the blood removed from the vein in the arm all yielded staphylococcus pyogenes. A vaccine of 500,000,000 was prepared from the various strains.

January 10. She passed a very restless night, but there was a marked improvement in the quality of her pulse, which at 8 a. m. was 108, temperature 99; 12 noon, temperature 99.2, pulse 102; 4 p. m., temperature 98.4, pulse 108; 8 p. m., temperature 100.6, pulse 100.

January 11. At 3 a. m. the temperature rose to 103.4, but gradually dropped, ranging from 98.4 to 100.2.

January 13. The patient has been gaining in strength during the last 48 hours, and the temperature during this period has remained low, ranging from 98.4 to 99.2. The wound was dressed at noon, the packing at the knee was not disturbed, but the gauze from the mastoid and neck wounds was removed and the parts looked healthy. No pus was found in the wounds, but by gently pressing upward from the neck pus flowed freely into the mastoid from the bulb. Temperature, 6 p. m., 98.2; complained of feeling chilly. Temperature, 7 p. m., 104.

January 14. Temperature, 8 a. m., 100.4; 12 noon, 101.4; 4 p. m., 101.4; 8 p. m., 103.6. Wound dressed at noon; pus still flowing from the mastoid end of the bulb on gentle pressure on the neck. Gentle irrigation with a small syringe through the mastoid end of the bulb brings away small clots and foul-smelling pus. At 4 p. m., ½ c.c. autogenous vaccine was administered in the arm.

January 18. During the past three days, the patient's condition remained about the same, the temperature assuming a general septic character. Early morning temperature today, 98; 12 noon, 103.8; wound dressed at 2 p. m., both neck and mastoid wounds granulating. The ligature at the distal end of the jugular having sloughed off, a small canula was inserted in the vein, and with gentle irrigation a partially broken-down and foul-smelling clot, mixed with pus, was washed out of the mastoid end of the bulb. A gauze wick was introduced into the mastoid end of the bulb, and the dressings reapplied. Twelve minims of vaccine given in the arm at 4 p. m.; temperature, 104.2. At 8 p. m. the temperature was 103.4. The wounds were dressed every second day, owing to the difficulty in draining the bulb, which continued to secrete pus in considerable quantities. Notwithstanding the septic temperature the patient was running, she continued to gain in strength.

January 20. Twenty minims of vaccine were administered in the arm. The temperature continues irregular.

February 4. For several days past the child has complained of a pain in the left leg. An examination showed a decided red spot about the size of a 50-cent piece on the outer side of the left leg, which was tender and fluctuated on pressure. She was taken to the operating room and, under ether, the mastoid and neck wounds were dressed. Pus had practically ceased to form in and around the bulb, both wounds granulating rapidly. A free incision was then made over the swelling on the left leg, and a large quantity of thick, greenish pus, which had burrowed down between the muscles of the leg, was evacuated. The abscess sac was thoroughly curetted and the wound packed with iodoform gauze. The temperature which had been practically normal for several days, suddenly rose at 6 p. m. to 103.4. This dropped gradually during the night, and at 8 a. m. on the morning of February 5th was 98.2. From this time on, the temperature gradually became normal. The wounds in the mastoid region, the neck, and the leg, granulated rapidly, and the child's convalescence was uninterrupted, and she was discharged from the hospital on February 12. The subsequent dressings were done at the house and at my office.

To fully appreciate the septic character of the temperature in these cases, reference to the charts which are herewith appended is necessary. In Case I, the ordinary stock streptococcus serum was used. In Case II, a vaccine of 500,000,000 was prepared from various strains made from a culture obtained from the blood taken from the patient's arm and from the clots of the lateral sinus and jugular vein.

January 14. 1.20 c.c., or about 25,000,000 were injected into the patient's arm.

January 16. 12 minims were injected into the right thigh.

January 20. 20 minims were injected into the left thigh.

As there was no appreciable influence on the temperature, no further injections of the vaccine were given.

The leucocyte counts were as follows:

December 25. 13,000.

January 3. 16,400.

January 5, 13,100.
January 8, 12,000.
January 17, 11,000.
January 18, 12,000.

Relative Number of Leucocytes.

	Per Cent.	Number Counted.
Small lymphocytes	26.0	65
Large lymphocytes / Large mononuclears	7.6	19
Polymorphonuclears	66.4	166
	100.00	250

All of the bacteriological work and blood counts in these cases were done by Dr. J. B. Nichols, the pathologist of the Hospital.

While the infection in Case 1 was of a more virulent type, it did not present the same difficulties as were met with in Case 2.

An interesting point in Case 2 is, that in the original operation the sinus wall was exposed from just below the knee to near the bulb, and at about an eighth of an inch removed, exposing the sinus which at this time did not appear to be affected. On January 9, when the sinus was thoroughly exposed, it was found solidly thrombosed from near the torcular to and involving the jugular vein quite to its full extent. The point of infection apparently was at the knee, although there was no diseased bone in this vicinity. The infection must have taken place through some of the smaller veins rather than from any direct contact of septic matter with the sinus itself. The greatest difficulty we met with in the management of this case was in relieving the bulb of the septic clot, and its resulting perisinus inflammation. It was the prolonged suppuration in this region which caused the continued general septic condition.

It is the bulb complication that causes so much trouble in the management of these thrombosis cases. While I removed the bone covering the sinus as far down as the point where it bends, it was not sufficient to bring complete drainage, and

it was only by the most persistent and constant cleansing of these parts that I was able finally to overcome the suppurating condition.

Fortunately, both of these cases were brought to a successful termination, notwithstanding that a marked general septic condition was present before the source of infection was shut off from the general circulation.

How much the vaccine therapy had to do with the subsidence of the septicemia is a question. In the first case, I believe the antistreptococcus serum had a beneficial effect, but in the second case I doubt whether the autogenous vaccine had any effect at all upon the general systemic poisoning.

REPORT OF SPONTANEOUS CURE OF SIX CASES OF UNRECOGNIZED SINUS THROMBOSIS, ACCIDENTLY DISCOVERED DURING OPERATIONS.

By EWING W. DAY, M. D., Pittsburg, Pa.

There is probably no aural complication in which the chief clinical phenomena more clearly indicate the morbid changes present than a virulent infective sinus thrombosis. The symptoms which first attract attention, are the marked irregularities in temperature, intermittent in character and ranging from normal or sub-normal, to 103 or 105 and associated with chills followed by sweating. These symptoms are due to the periodic discharge of septic material into the blood stream, as a result of the breaking down of the infected clot. The presence of a bacteremia with later evidences of secondary foci of infections, complete the cycle of symptoms generally considered necessary for the diagnosis of infective sinus thrombosis.

For several years the writer has been slowly forming the opinion, that there is a far greater number of sinus thrombosis from aural infections without these marked symptoms than is generally supposed.

The steps in the typical sinus thrombosis are:—

First. The original ear infection.

Second. The extension of this infection to the inner wall of the sinus, and clot formation.

Third. The breaking down of the clot.

Fourth. A free discharge of the broken down material into the general circulation.

Necessarily the absence of the fourth step must produce marked changes in the classical symptom otology.

From personal clinical findings, we would classify sinus thrombosis into three general groups:—

First. Those in which the septic material is drained directly into the veins, or aspirated into the opposite sinus.

Second. Those in which the distal ends of the clot remain firm and become organized, the center breaking down and draining through a rupture of the sinus wall.

Third. Those in which the entire clot becomes organized and sterile, with obliteration of the sinus.

In forty-five cases of thrombosis of the lateral sinus, we found thirty-six belonged to the first group; three in group two and six in the third group, giving nine out of forty-five cases or 20% atypical cases for our series.

All of the three cases of the second group had a perforation of the posterior mastoid wall over the sinus, the rupture of the sinus taking place at this point and discharging into the necrotic mastoid cavity. The symptoms were not distinctive or severe, resembling those of a mastoiditis with an epidural abscess. All made uneventful recoveries. Dr. H. Friedenwald at a meeting of the L. R. and O. Society, 1913, reported in detail two cases of this kind where the symptoms were more severe.

It is to the third group in which the clot becomes organized with obliteration of the sinus that I wish to call special attention. Of these we have found six cases, the diagnosis, made on the operating table was not previously suspected. How many have been passed by unrecognized, will never be known. Their previous clinical histories are incomplete and many important symptoms were probably not noticed. The time of the formation of the clot can be only conjectured.

Case I. A man aged 52, seen in October, 1910, with history of several attacks of A. O. M. P. in previous years. In March 1910, had an A. O. M. P. complicating LaGrippe. The discharge was free, but early in the attack, the patient suffered from severe pain in the ear and frequent spells of dizziness. Occasionally there was vomiting, but there was no history of high temperature, chills or sweating. The ear discharged for one month. Since then there had been no discomfort referable to the ear, but the patient complained almost constantly of some headache on the right side. Early in October the headaches had increased in severity and sleep could only be obtained by opiates. With the increase of

pain, an irritable temper and mental hallucinations developed. Examination of the ear showed no discharge. The tympanic membrane was intact, but injected. There was one point of slight tenderness about one-half inch below and behind the mastoid antrum. X-ray showed right mastoid to be opaque. Blood examination: W. B. C. 18,800 Polys. 80%, Lymphocytes 17%, Large Mono, and Transitional 3%. Several temperature readings varied 0.2 degree above and below normal. Caloric tests were positive. Brain abscess was suspected.

On operation, the mastoid was sclerotic to a medium degree, no pus or necrosis being present. The cerebellum was uncovered, the sinus found yellow in color, and normal in size, but obliterated by a firm fibrous tissue, removal of which was attempted with only partial success.

The healing was rapid, and the headaches and abnormal mental condition disappeared. Whether this removal of the fibrous tissue, or the decompression over the sinus from removal of the bone produced relief is conjectural.

Case II. Was a Negro preacher in middle life, who applied for treatment at the Eye and Ear Hospital, for a profuse purulent otitis in the left ear. He gave a history of having contracted measles, two months previous to admission, followed by earache and profuse discharge which continued without decreasing in amount. Admitted to the hospital on November 28, 1909. Profuse purulent discharge from left ear. On December 14th, the discharge was still profuse, with severe pain in left temporal region, and tenderness over the mastoid. Smear of pus showed pneumococci. W. B. C. 17,400. Polys. 87%. Lympho. 7.6%. Large Mono and Trans. 4.4%. Eosinophiles 3%. Mast cells .7%. Temperature for previous sixteen days at no time above normal and no chills. Mastoidectomy under local anesthesia. Large mastoid filled with granulation and pus. Perisinus abscess over the knee of the sinus. No granulation over the sinus. Sinus opened and found filled with a firm clot, a portion of which was removed, showing signs of becoming

organized. Laboratory report showed sterile clot with beginning formation of connective tissue.

Case III. Report of attending physician—Female, age 22. Had usual diseases of childhood with C. P. O. M. at intervals since 6 years of age. On October 1st, 1914, was attacked with acute pain in right ear. During night patient was chilly with fever, ranging from 101 to 104 degrees, headache and delirium, which continued for one week, when delirium and ear symptom subsided. After second week the headaches disappeared, but on three different occasions the patient developed a cyanosis, lasting for two or three hours. The patient continued to run an irregular temperature, 100° to 103°, for four or five weeks, with a slight discharge from the ear. No mastoid tenderness, but tenderness along the anterior border of sterno-mastoid muscle. Widal test was negative but the case was considered as a probable irregular typhoid. The patient, in apparent good health, consulted me December 18th, for the C. P. O. M. Middle ear was filled with a mass of granulations and foul pus. A radical mastoid was advised. This was done one month later. The mastoid, when opened, showed a cavity filled with granulations. Nature had done a partial Stacke, and through this, granulations crowded into the deep external canal and middle ear. The posterior mastoid wall was absent and the sinus was covered with granulations, when exposed in the mastoid cavity. The sinus was uncovered for two inches. It was of a yellowish color, hard and tense. A small exploratory incision showed it to be totally occluded with organized tissue. The sinus was not further molested. The usual Stacke operation was completed and the mastoid incision closed. The cavity was completely epidermatized and dry in five weeks.

Case IV. A girl of 5 years, contracted scarlet fever in January, 1913, complicated with a double A. P. O. M. The scarlet fever was of a severe type with high temperature, reaching 106° on two or three occasions, but no systematic record seems to have been kept. If chills or sweating were present, they were not noticed. Admitted to Eye and Ear

Hospital on March 7th. Both mastoids tender, marked swelling over left. Patient was emaciated and showed all signs of a severe illness. Blood examination: Leucocytes 9800. Polys. 67%. Small Lymph. 23%. Large Mono. 8%. Eosinophiles 1%. Transitional 1%. Temperature 101°.

The outer table of both mastoids lifted off as a sequestrum, interior infiltrated with foul smelling pus, but with little bone necrosis in the right and small necrotic area in the left, exposing the sinus, over which was a small perisinus abscess. The sinus was uncovered downward and posteriorly for 6 C. M. Had a dull yellowish appearance and was firm to the touch. There was a small gangrenous area, where it was exposed in the mastoid cavity. Sinus was incised at this point and found filled with an organized clot. Some of this was removed for examination. The remainder was not disturbed. On the second day pain and tenderness developed in left wrist, lasting for two weeks, but disappeared without pus formation.

The laboratory report showed a few streptococci from pus smears in right mastoid. Smear of pus from left mastoid was negative. Cultures from thrombus of left lateral sinus negative. The healing in this case was very slow, extending over four months. The temperature being always 100° or less.

Case V. Paul P., age 7 years: had had a discharging ear since infancy. Was brought to the Eye and Ear Hospital for an acute exacerbation, with marked post auricular swelling, April 7th, 1915. Smear showed the usual mixed infection of C. P. O. M. No blood examination made. Operation: Mastoid necrotic, large quantities of cholesteatoma. Necrosed bone over sinus removed. The sigmoid sinus, yellowish in color and firm to the touch. The lateral portion of sinus, from the entrance of the superior petrosal sinus back towards the torcular, normal in color and contained fluid blood. An exploratory incision in sigmoid sinus, showed complete obliteration by organized tissue, probably recent.

The day following the operation, temperature reached $101\frac{2}{5}$°, receding to normal on second day. Discharged from

hospital on eighth day to be attended by a specialist in a neighboring town, who reports the mastoid wound nearly healed. The mother gave no history of a previous illness, other than ordinary ailments incident to childhood.

Case VI. Was a child of about 8 years, operated on by my associate, Dr. Waldie. The history of this case has been misplaced at the hospital and could not be found and this outline is given from memory. The patient was admitted to the hospital for a C. P. O. M. with post auricular swelling and tenderness. The usual conditions were found in the mastoid with the sinus presenting through a perforation of the posterior wall. The sinus was freely exposed and incised and was found occluded by organized tissue. The case was treated as an ordinary mastoid and kept in the children's ward until completely healed.

The most striking characteristic of these cases is, first: that absence, as far as could be ascertained, of pyemic symptoms, generally considered so necessary an accompaniment of infective sinus thrombosis and the defensive efforts of nature. It seems reasonable to suppose that they were all caused by an infective organism, as five had necrotic mastoids adjacent to the sinus and Case 1, had a history of several attacks of A. P. O. M., the last one being several months before the sinus condition was discovered.

The absence of a history of high temperature and chills is marked in five cases. The chart of Case 3, at the time of the supposed typhoid attack in connection with an active pus secreting ear, would suggest a sinus thrombosis to an otologist, but this infection must have been of a mild or attenuated nature, or the clot would have broken down instead of organizing. Case 2, was under hospital observation for fourteen days prior to operation, with practically a continuous normal temperature. The clot in this case was evidently of recent origin.

In case 4, the high temperature reported as having occurred during the scarlet fever may have been in part, due to the unsuspected thrombus, though high temperatures are frequent in severe scarlet fever.

Diagnostic symptoms, referable to the sinus condition, seem to be absent previous to and at time of operation, with the possible exception of Case 3. In Case 1, the early symptom was severe temporal pains, with mental hallucinations, while the other four had only those general symptoms common to sub-acute mastoid.

In these cases the condition present was not suspected. In Case 1, the exploration was started for a supposed abscess. In the other five, the sinus was already exposed in the mastoid cavity and further operative exposure revealed what had taken place.

The slight interference with the contents of the sinus, seemed to have had no influence in the complete recovery of the patient.

In Case 1, where firm connective tissue had formed, its removal was attempted with only partial success, and I believe it was meddlesome surgery. In two others where the clot showed partial organization, a portion was removed for laboratory examination. In the remaining three, the centers of the occluded sinuses were incised and on finding well organized clots, were not further molested.

Nine atypical cases of sinus thrombosis out of forty-five, or 20%, would seem to justify the conclusion, that absence, of classical symptoms does not justify an assumption of no thrombosis. The fact that in 13½% of these cases, nature effectually cured the condition before its discovery, would either indicate that this obliteration of the sinus occurs more frequently than generally supposed, or that it was our fortune to have an unusual number; that when it does occur, the infection is probably attenuated or of low virulence, creating slight constitutional disturbances; that the presence of pyemic symptoms indicates an active virulent organism, with great resistance to nature's curative action, producing symptoms that are seldom overlooked and always demand operative interference. In milder cases the diagnosis is rarely made and when discovered, nature has already made the cure.

Discussion.

Dr. James F. McKernon, New York City, had had three cases somewhat similar in character to those reported by Dr. Day. In all these cases the otitis media had developed not weeks but months before, and they did not present themselves for operation until a long time had elapsed since the beginning of the lesion in the ear. All had chills and fever, the fever going as high as 106 degrees in one case. The patients did not present themselves on account of the running ears, but because of the tenderness and edema which had suddenly appeared behind the ears. The mastoid in each case was filled with granulation tissue and foul-smelling pus. Two had occurred where one would not expect to find sinus thrombosis; in the third there was a distinct organized clot. All were treated in the usual manner, and all made an uninterrupted recovery. In a fourth case which he had seen in consultation the mastoid operation was performed by a colleague. There was a firmly organized clot, so far as macroscopic appearances were concerned. The colleague decided to open this clot. He removed some material for bacteriological culture. The case did not do well, and the patient subsequently died from empyema. Autopsy was not allowed, but death was probably due to encephalitis. He believed it was better to leave the clot alone. He did not believe that septic sinus thrombosis ever occurs without fever, though the history of the fever might not be elicited.

Dr. E. B. Dench, New York City, alluding to Dr. Bryan's second case, in which the essayist said he did not expose the sinus further because, after opening a small distance, the structures seemed healthy, advocated making a good, full exposure of the sinus in all cases when the sinus was either accidentally or intentionally exposed. The small vessels absorb a certain amount of pus from the area of infection in the mastoid. When the sinus walls swell, there is a tendency for this septic material to be carried from the external to the internal wall of the sinus, and to cause a parietal clot,

Some of the most severe symptoms follow the formation of a parietal clot. Referring to Dr. Day's report, he had seen two similar cases. In one case nothing was found but a cord. Nothing was done, and the patient made a complete recovery. In the second case the patient, a man with acute otitis media, had been under the care of a physician in New York City, who had done a myringotomy. The speaker saw the patient four weeks after the myringotomy, at which time there was post-auricular swelling. When he opened the mastoid he found the inner table completely destroyed the sinus wall necrobe and the vessel occluded by a clot. He resected the jugular. The jugular wound healed perfectly. Just before the patient was to be dismissed from the hospital, he suddenly developed a temperature, and died. Some of the clot taken from the bulb was sent to the laboratory and found to be perfectly sterile. The clot from the torcular end of the sinus was also sterile.

Dr. John F. Barnhill, Indianapolis, Ind., thought these cases quite remarkable, and indicated very clearly that all had not yet been learned about mastoiditis and its complications. He had never seen a case such as Dr. Day had reported. The study of the temperature during any period of mastoiditis was so essential that he always preferred to have a good nurse in charge, or to have the patient in a good hospital, in order that the first indication of sinus infection could be promptly noted. In all cases with temperature above 102 degrees he suspected some complication; when it went above 103 degrees he knew something was wrong, and a temperature of 103½ to 104 degrees indicated serious complication. In his experience uncomplicated mastoiditis had seldom run a temperature over 102 degrees. In the presence of other disease the temperature might not be due to the mastoiditis at all. He had recently had a case in which the temperature was due to malaria, and yet the patient had all the classical symptoms of sinus thrombosis. With reference to uncovering the sinus, as had been done at the first operation, he thought that should be done if the

temperature still indicated infective sinus thrombosis. He did not believe one could tell that the sinus is diseased from the outside in the early stages of the affection; it was necessary to open the sinus to determine the presence of an infective clot. The procedure followed by Dr. Bryan in the first case, of curetting without ligating the jugular, was not dangerous because he did not go to the bottom of the clot. In his opinion, as soon as it is found that there is a clot in the sinus, one should stop the operation above, go below, and ligate the jugular, or remove the jugular, as may be. Occasionally it was difficult to determine whether or not to open the sinus. He had recently had a case in point. The mastoid had already been opened incompletely twice. On the occasion in question he came upon a large quantity of pus, not only in the tip of the mastoid, but in the neck. He uncovered the sinus, which seemed to be all right. He waited two days, two other chills occurred, and the temperature went to 105 degrees. Pus in a wound would not give a temperature of 105 degrees; in such case the sinus must be looked into, as was done; the sinus contained pus, the jugular was excised, with rapid recovery as a result.

Dr. J. A. Stucky, Lexington, Ky., said Dr. Day's paper raised the very important question of the symptomatology of sinus thrombosis. In the South the opinion prevailed that in the East otologists open the jugular when it is not necessary. The presence of temperature alone was no indication for opening the lateral sinus; but temperature with chill, especially in an adult, should lead one to be very careful. He was quite sure that he had had cases of sinus thrombosis which he could not operate that had recovered. He cited a case in which a colleague wanted to do a secondary operation, in which there was every classical symptoms of sinus thrombosis, but in which the family would not allow further operation. After two or three doses of castor oil resulting in free purgation the patient rallied and recovery followed. It was contended that the colleague wanted to do an unnecessary operation, and therefore court proceedings

were threatened. However, in the presence of high leucocytosis, temperature and chill following a mastoid operation the sinus should be opened.

Dr. John B. Rae, New York City, acknowledged the value of the report of Dr. Day but held that the cases cited raised no question as to the methods of procedure in the treatment of sinus thrombosis with symptoms. These were all old cases. Even in those in which the infection appeared to be new, it was probably old, for at operation a well organized clot, a firm fibrinous clot, was found. It would be hard to associate an organized clot with a very recent case.

Dr. George L. Richards, Fall River, Mass., thought cases such as those reported by Dr. Day were probably slow cases, in which the clot was formed, and then walked off by Nature. This might occur with a typical up-and-down temperature.

Dr. B. Alexander Randall, Philadelphia, fully concurred with Dr. Rae in the opinion that the process was not a new one, yet it was probably not due to chronic suppuration. Almost annually for a dozen years and more he had been insisting on the self-limiting trend of the acute in contrast with the septic course of the chronic cases. One case in Dr. Day's notable series sounded like a chronic suppurative condition; but that was also a recurrent condition, and the history would indicate to his mind a recurrent acute attack. He thought one missed something of the pathology of these cases in omitting the word *phlebo*-thrombosis. One had to deal primarily with phlebitis, the thrombosis being secondary. Unless it broke down and gave rise to secondary complications, the thrombosis was the less important matter. He gravely doubted whether, in the presence of a septic clot, there was any formation of fibrous coagulum and occlusion of the vessel. He thought it possible, on the other hand, for a condition of this kind to occur and to pass through the stage of thrombosis, and for Nature to re-establish the circulation. He had never met the cord-like sinus and occlusion

of the vessel in his septic cases, although the jugular is often thus found. This was not because he had not laid bare the sinus, for he had done this very extensively in a large number of cases. He had reported one case at least, which went on to complete blindness, pointing back to a healed sinus thrombosis.

Dr. Emil Amberg, Detroit, Mich., cited a case previously reported elsewhere, in which the general practitioner thought there was sinus thrombosis judging per exclusionem. He operated, found pus in the antrum, sinus thrombosis, ligated the jugular, opened the sinus, and got some bleeding from below none from above. Three days later the thrombus reaching to the Torcular Herophili was removed. The patient recovered.

The credit for the diagnosis in this case belonged to a great extent to the general practitioner.

Dr. G. H. Powers, Jr., Boston, Mass., relative to the question of symptoms, cited a case in which the patient, a little girl, seen by him in the Out-Patient Department of the hospital, gave a history of earache for some weeks. She had wry-neck, temperature of 100 degrees, leucocyte count of about 15,000. She had been making visits every other day to the family physician. At no time did the temperature go over 101 degrees. The mastoid was opened, and found to be comparatively normal, except one cell. He opened the sinus, and found brown pus. The patient had enlarged glands in the neck, infection of which was due to the thrombosis, and which probably accounted for the wry-neck. He cited two other cases in which the mastoid was normal, the sinus involvement resulting directly from infection in the middle ear through the jugular bulb.

Dr. E. Terry Smith, Hartford, Conn., thought the only positive symptom in sinus thrombosis was the temperature. The blood count was entirely unreliable.

Dr. Sargent F. Snow, Syracuse, N. Y., had tried autogenous vaccines in a number of cases of streptococcic in-

fection, with practically no favorable results. He cited in detail a case of recovery following double mastoid operation, ligation and resection of the jugular of one side for sinus thrombosis, with very high fever and evidences of meningitis. In this case, as in the one cited by Dr. Stucky, the turning point seemed to follow the clearing out of the bowels.

Dr. Frederick L. Jack, Boston, Mass., said the discussion had left the impression that the subject of sinus thrombosis was being discussed for the first time. He judged, from what had been said, that one might as well leave these cases to nature, inasmuch as some die and some get well. His own idea on the subject was that if, at the mastoid operation, sinus thrombosis be found, and if there had been little or no temperature, and no chills, then, whether the mastoiditis be acute or chronic the sinus should be left alone. In the presence of these symptoms, the clot should be cleared out. High temperature at the time of operation and the presence of grey pus were indications for the clearing out of the thrombus.

Dr. George E. Shambaugh, Chicago, in support of the report of Dr. Day, called attention to the fact that in recent years several cases have been reported where the thrombosis of the sinus existed without causing any rise in temperature.

Dr. Harris P. Mosher, Boston, asked whether Dr. Day had had any difficulty in locating the thrombosed sinus. He had had a case in which he experienced great trouble in locating the sinus. He went back and forth over the locality, but got no evidence whatever from palpation, and finally opened the lateral sinus. Even then he could not find the thrombus.

Dr. Frank T. Hopkins, New York City, cited a case in which there were complications. The patient, a child, had had scarlet fever, discharging ear, and acute mastoiditis. The mastoid operation was performed without special note, but five days afterward the symptoms of sinus thrombosis.

the sinus was opened, and the jugular removed. The child seemed to be doing fairly well for a few days, when the temperature rose, and the termination showed that there was infection toward the bulb and toward the torcular. There was nothing at the time of the operation to show infection of the sinus in the portion toward the bulb, but culture from a smear taken from the part toward the torcular showed infection in that direction. The temperature again shot up. A culture made from a smear taken from the portion toward the bulb was again negative. He curetted back nearly to the torcular. The patient recovered. He mentioned the case as showing that the infection continued even after removal of the jugular, even after the removal of bone down to the lower portion of the sinus channel, the so-called Goünert operation. The infection was present in the posterior portion of the sinus, and the child did not improve until this was cleared out back toward the torcular.

Dr. Bryan in closing the discussion, said he did not advise uncovering the sinus. In the first case the sinus wall was not removed; in the second case it was opened accidentally and the opening somewhat enlarged in order to inspect the sinus. From outward appearance it was not an infected sinus. Blood cultures were made. There was a question as to the efficacy of douching in such cases. In his cases the patients were suffering intense pain, and douching relieved this; it also gave freer drainage in consequence of the liquefaction of the pus. Referring to the matter of curetting from the sinus end of the bulb in order to ascertain whether there was occlusion, he thought it better to gently remove the occlusion by removing the clot.

Dr. Day, in closing the discussion, was not unmindful of the fact that a paper of the kind he had presented was a curiosity more than anything else, and that harm might come out of it. The conservative individual might be led to say, "Leave the sinus alone"; the enthusiast might say, "Of course, uncover every sinus." He had long believed that there were more cases of sinus thrombosis than had been

suspected. He could not agree with Dr. Randall with reference to phlebitis. Was it to be supposed that, with so many cases of mastoiditis as occur, the infection stays right there and that nature takes care of it? He believed that it formed a clot in more cases than might be suspected, and that nature was able to sterilize the clot before it broke down. Otherwise how could one account for the cases in which the ends of the clot are sterile? He agreed with Dr. Rae that the cases are chronic when the otologist gets them. He believed with Dr. McKernon, that the sinus should be opened. No harm would come from opening the sinus. In substantiation of this, he cited a case in which, at autopsy, it could not be detected where the incision had been made.

NERVE DEAFNESS AND MUMPS.

By THOMAS HUBBARD, M. D., Toledo, Ohio.

The statistics of collective investigations of deafness due to mumps impress the importance of more careful study and clinical observation of this infectious disease.

Most of these reports are institutional and they are presented to call attention to the fact that this type of nerve deafness is not so rare as would be inferred from text books, and that there is a wide discrepancy between deaf mute statistics and current clinical records. It is rather surprising to learn that mumps is given as the cause of nerve deafness in three to five per cent. of the cases of deaf mutes. There are about 50,000 deaf mutes in the United States (41,000 in 1890) and of these one or two thousand are mump cases. In fact mumps rank close to scarlatina as a cause of absolute bilateral deafness, and probably the ratio is more favorable to mumps where unilateral nerve deafness is considered. Huebner, Goetingen Clinic, ascribes to mumps two per cent. of the cases of labyrinthine deafness. In contrast to this statement attention is called to the fact that the most comprehensive clinical report published is that of Dr. G. W. Boot (7) who gives an analysis of only 51 reported cases. This must be taken as an indication of indifference and in part explains the reason why the pathology has received scant attention. It stands as an opprobrium to our special art that the essential nature of this disease process cannot be expressed in more precise pathologic terms. Theories are beating around the bush and may scare up something, but rational treatment and effectual prophylaxis must remain in a comparatively hopeless confusion until the pathology is better understood.

Clinical Features. Auditory symptoms may appear any time during the first week of mumps or they may be deferred until two weeks after initial symptoms. In the latter condi-

tion relapses and late involvement of other glands with obscure local symptoms must be taken into consideration. Labyrinthine symptoms have been known to appear several days in advance of any gland symptoms, and in a few cases the typical symptoms of mumps were entirely overlooked. Tinnitus is the most common symptom of labyrinthine invasion and it is usually followed by vertigo, nausea, vomiting, and disturbed equilibrium. Unconsciousness, variable fever and headache are observed in some cases.

The destructive process may be so rapid that all of these ear symptoms are wanting, absolute deafness occurring in a short period or during sleep. In the majority of cases both cochlear and vestibular end organs of the auditory nerve are simultaneously involved and after a variable period of inflammatory excitation are functionally destroyed. A comparatively few cases exhibiting marked symptoms recover a useful degree of functional activity. Occasionally this can be definitely determined by discovery of "islands of tone perception" in certain registers, and in more favored cases "islands of deafness."

In considering the relation of mumps to labyrinthitis we must include the unusual types of the disease. The organs commonly involved, either simultaneously or consecutively, given in order of frequency, are parotid glands, submaxillary glands, sub-lingual glands, testicles, ovaries, mammary glands. To these should be added meninges and internal ear. Clinical records give evidence that the disease may primarily involve any of these organs and the characteristic clinical picture may thus be decidedly obscured or very late in appearance. For instance, absolute nerve deafness, single or double, may appear and later some of the symptoms usually considered secondary—such as orchitis—may aid in clearing up the etiology of the loss of function of hearing. Or the primary symptoms of gland infection may be entirely overlooked and days or a week or two later labyrinthitis may be discovered.

The susceptible age has a wide range and this gives rise to two confusing factors in diagnosis. In young children

subjective symptoms and even deafness in one ear may not be mentioned, and attendants are quite liable to overlook casual suggestions of such symptoms as tinnitus, vertigo and nausea in young children. Co-incident gastric disorder and symptoms referable to fever and meningeal irritation may lead the medical attendant off the track, and it is not uncommon that the disturbed equilibrium and even the deafness are discovered some time after the child has recovered. In adults a mild attack of mumps may be treated with as little concern as an ordinary tonsillitis, or a metastasis or mixed infection may dominate the clinical picture to the degree of obscuring all ear symptoms.

All of this leads to confusion of clinical data as to the time of auditory involvement; and further it offers a suggestion that some of the obscure cases of partial nerve deafness encountered in aural practice might be traced to mumps if careful inquiry be made. The cases having only a mild or aborted labyrinthitis are precisely the ones that would escape detection during the acute attack, especially in those having severe general symptoms or an active metastasis to such organs as testicles or ovaries. Mumps as a cause of hypoacusis of obscure origin deserves careful study by otologists.

Mixed infection may occur in mumps and account for the so called virulent type. Meninges, pleura, pericardium or peritoneum may be involved and most serious complications or lasting sequella result.

Case Report. O. Mauthner (3). 1. Female, 37. Recently took mumps from her children. Had violent fever and both parotids swelled. On the very first day of the disease the mother heard a ringing in the left ear which became louder until a persistent tinnitus was present. Hearing not disturbed until the third day, when she noticed that she could not hear on left side. No pain in the ears. Forty-eight hours after loss of hearing violent vertigo developed, with a little nausea. The vertigo reappeared next day in milder form and so on each subsequent day. For eight days there was double vision and for three weeks disturbed gait. Treatment was unavailing. Not until after six weeks did all the

symptoms vanish, aside from deafness and tinnitus which still persisted after eight months.

Examination at this time showed eyes wholly normal and no disturbances of motor cranial nerves. Normal drumheads, both sides. General condition good. The hearing was tested by all methods. The excitability of the semicircular canals remained although somewhat depressed. Standing and walking were not entirely normal. Frequent effort to show the presence of spontaneous nystagmus failed. The diagnosis was unilateral deafness with retained excitability of the vestibular nerve. The author (O. M.) believes this case was toxic in character, an acute toxic or infectious neuritis due to a general infection. Influenza may produce the same lesion, without involvement of the middle ear, but with vestibular symptoms. In a case of Wittmaack's sudden influenza deafness was followed in two weeks by marked aural vertigo. The left ear was totally deaf but the right ear improved under treatment. Tinnitus was also persistent in this case. In another case there was a high degree of bilateral deafness and tinnitus but without vertigo.

Other cases of the so called toxic type will be mentioned in passing.

Hegener described an acute bilateral rheumatic neuritis acustica, which may be supposed to stand in near relation with influenzal neuritis. The patient had charge of a cellar which was damp and developed rheumatic pains in the knees. Sudden tinnitus, which did not abate, set in, together with spinning vertigo and nausea, later violent vomiting. In a few days the fever and vertigo had subsided, but there was unilateral deafness and tinnitus. Similar phenomena have been known to result from erysipelas.

H. Haike, case report (9). Male, aged 19. Student. Taken ill March 24th, 1910, with malaise, slight fever and sudden impairment of hearing on the right side, with no pain. Nothing abnormal in membrana. Absence of bone conduction and lateralization. No vertigo; pupils normal; fundas oculi normal. On March 27th (three days after deafness) a double orchitis began, with stepladder tempera-

ture rise reaching 40.4 deg. C. on the 29th. Two days later there was a critical drop to 35.5 deg. C. Vomiting at that time was thought to be due to a reflex from the testicle.

The author did not see the case until about four weeks after its onset. The tube was pervious but the voice and watch were inaudible and likewise the tuning fork C to C^4 air conduction. Bone conduction for the deep C was shortened. Diagnosis, acute labyrinthitis. The patient had lost 20 pounds and was in very bad condition. Three days later complete hearing tests were made, and audition was present in some degree to certain tests. This, however, represented only a remission as five weeks later and subsequently the condition found was almost total deafness. The vestibular nerve was not permanently affected. It is probable that the primary deafness was due to an exudate, which then underwent absorption with some improvement of hearing. The final deafness was complete save for a "tone island" in C^2-C^3. The absence of vertigo leads us to conclude that the lesion was retro-labyrinthine neuritis of the acoustic nerve. There was a general infection and the supervention of orchitis naturally suggests mumps deafness. Parotitis was absent, however. Grandenigo once reported the following: A boy aged two and one-half developed mumps, and his brother, two years older, was taken later with orchitis but not with mumps. On the next day the latter lost his hearing —both ears—completely and never recovered it.

The connection between the parotid and testicle in mumps is so obscure that Huebner believes the only way to explain the involvement of the latter is by direct inoculation of the urethra with virus from the salivary glands. This is not necessarily Huebner's personal opinion but the view which obtains among military surgeons. The mechanism by which the labyrinth is affected in mumps has been much debated. The fact has been published that the labyrinthitis has sometimes preceded the parotitis by several days, this sequence being by no means rare. All theories thus far advanced fail to explain the association of the two lesions. The most natural supposition is this: that mumps is a general infection

in which the parotids, labyrinth, testicles and ovaries and other organs are involved in varying degrees of severity. The labyrinth and testicles alone present the permanent lesions and this will be further considered.

Sudden deafness in children, almost always bilateral, may also occur without parotitis, etc. In these cases there is sometime meningeal irritation with disturbed sensorium, which also at times accompanies mumps. Professor Voss believes that these cases are best understood as abortive parotitis—that is primary parotitis with local symptoms so obscure as to escape detection, and it is possible that in such cases we have clinically an exclusive localization of the same process in the internal ear.

Author's Case Reports. Labyrinthitis caused by epidemic parotitis.

(1) L. H. Boy, aged 13, son of a physician. Had epidemic parotitis a few weeks previous to consultation. He was considered quite ill for a day but had no special symptoms sufficient to call attention to the ear. The parents are positive that the hearing was normal prior to this sickness. A few weeks after recovery the mother noticed inability to locate direction of sound. This was the first suggestion of deafness, but on close inquiry the boy distinctly remembered attacks of dizziness during the one day's confinement in bed and later vertigo on standing. Membrana tympani normal. Medium sized adenoids were removed and later functional tests were completed. Hearing in the left ear normal. Right ear deaf to all tests.

(2) O. M. Boy, age 17. Labyrinthitis caused by epidemic parotitis. Two years previous had mumps involving parotids. He was not confined to bed. The subjective symptoms then noticed were tinnitus and vertigo and soon after absolute deafness. The tinnitus has persisted up to the present time. Total destruction of cochlear and vestibular function.

(3) V. C. Man, age 30. Double labyrinthitis caused by epidemic parotitis. Sudden deafness came on during an attack of mumps some months previous. General symptoms

not severe. Tinnitus and vertigo were the first symptoms and dizziness soon became so pronounced that for some time he could scarcely stand erect, and would fall if he turned quickly. At this time deafness is not absolute. He can hear only the loud fork in middle register. Sense of equilibrium is good. No response to recent inquiry, but I am informed by his physician that the deafness persists.

Diagnosis: Labyrinthitis due to epidemic parotitis. Cochlear function decidedly impaired, vestibular nerve involved but there is partial restoration of function.

(4) G. H. Boy, age 14. Labyrinthitis caused by epidemic parotitis. All of the male members of the family had mumps several months previous, and this patient, a rather feeble and poorly developed boy, was very sick, having been confined to bed more than two weeks with fever, headache, tinnitus and later disturbed equilibrium which persisted for some time. Total deafness in left ear. Vestibular reaction very sluggish. Membrana tympani and tube normal.

Diagnosis: Labyrinthitis caused by epidemic parotitis.

In addition to these cases I have records of several in whom there is well grounded suspicion that partial deafness was caused by mumps. One, a young man, volunteered the information without suggestion that his hearing was defective since an attack of mumps. Functional tests showed partial nerve deafness in one ear only. In another, 12 years of age, there is absolute nerve deafness, unilateral, the hearing being very acute in the other ear and nasopharynx and tube normal. The date of onset coincides with the attack of mumps which he had when very young. There is no other cause determinable.

A third case illustrates the necessity of routine inquiry along these lines. A switchman about 25 has absolute nerve deafness of the right ear. The other ear is normal as to hearing and both membrana tympani and tubes are normal. Inquiry failed to suggest any cause — infectious fever, injury, or suppurative inflammation. His mother was questioned later and she stated that he had had mumps when about six years of age and the defective hearing was noticed

soon after when he entered school. (Compare this with case No. 1, son of a physician in whom deafness was not noticed until three weeks after parotitis). This is the type of mumps labyrinthitis which is, I think, often overlooked. The indifference toward the disease as a transient infection, little feared as compared with other exanthema combined with the inclination on the part of the child to conceal a subjective symptom which if mentioned would bring a doctor and prolong the restraints of illness. All this delays and ultimately confuses the positive diagnosis.

Conclusion. The fact which I desire to emphasize is that, having excluded acute infections, typhoid, meningitis, etc., as well as traumatism and destructive suppurative otitis the circumstantial evidence will not rarely lead up to typical or obscure epidemic parotitis as the primary cause of labyrinthitis.

In an article in The Journal Experimental Medicine, September, 1909, on Experimental Parotitis, Dr. Isabella C. Herb, Chicago, gives results of personal investigations and an elaborate analysis of the reported bacteriology of mumps. The isolation of a diplococcus identical with that previously described by Laveran and Catrin and others, and which produced all of the essential lesions of mumps in dogs, would seem to definitely settle the question of the etiology of mumps, and throw some light on the pathology. One essential feature is that this is not a pyogenic organism. Suppuration rarely occurs in true mumps, and yet the inflammatory symptoms are often very severe. It explains the clinical fact that the system can tolerate such a very general dissemination of the infecting agent ranging from a meningitis to orchitis, and yet there is usually spontaneous and very rapid subsidence of symptoms after a brief period of active infection. The only serious functional sequellae, barring cases of extensive metastasis with mixed infection and general sepsis, are those following orchitis and labyrinthitis.

This aids in clearing up the pathology of mumps and especially the apparent selective action on the internal ear. The

infecting agent may be thoroughly disseminated through the system. It is highly inflammatory but non-pyogenic in action. This probably explains the clinical fact that fever is not a prominent or persistent symptom. It runs a brief course and we can ascribe its so called selective action to lack of resistance to invasion on the part of certain glandular tissues *together with peculiar anatomical construction.* In both labyrinth and testicle we find delicate tissue formation *in unyielding capsules.* Even non-pyogenic inflammation causes swelling and increase of tension within the firm capsules and it is scarcely necessary to assume that there is hemorrhage or a very copious serous exudate to account for the impairment or destruction of function of such delicate end organs as we find in the labyrinth — the organ of Corti, the thin membraneous partitions, the terminal vestibular fibres, the integrity and function of all absolutely dependent on perfectly balanced tension of the endolymph and perilymph. As a matter of fact mumps orchitis is not usually followed by complete functional destruction but there is sufficient clinical evidence of atrophy of the organ to verify the prevalent opinion that this type of orchitis does leave serious and occasionally permanent functional impairment. But in the labyrinth, as a rule the lesion is positive and permanent.

Politzer says: "It must be brought to notice, in considering the frequency of disturbance of hearing, that the auditory nerve possesses the greatest impressionability of all of the cranial nerves — that is, its function is much more often arrested by general diseases and chemical changes in the blood during infectious diseases, than the optic, gustatory, olfactory and sensory nerves."

The point which I wish to emphasize in this connection is that destructive hypertension is quite peculiar to labyrinthitis as compared with other special sense nerves within the cranium, and that compression is the destructive factor. The ductus endolymphaticus is the safety reservoir of the endolymph of the labyrinth and the very nature of its anatomical construction suggests the explanation that the destructive effect of hypertension due to inflammatory processes

within the labyrinth may thus in a measure be safeguarded.

The toxic neuritis theory, as applied to mumps, seems quite untenable. The absence of definite neuritis of other nerves of special sense, as well as of those nerves directly in contact with the infected glands, such as the facial, the submaxillary ganglion and the auriculo-temporal group, tends to discredit the toxic theory. The close anatomical association of facial and auditory nerves—the former intimately in contact with the parotid gland—might suggest an ascending neuritis and direct infection of the auditory nerve, but the clinical fact that labyrinthitis, or auditory neuritis is not peculiar to the parotid type of mumps does not support this theory.

Treatment. Labyrinth lesions of this type are almost comparable to traumatism in point of suddenness and period of warning. In the literature examined no brilliant abortive or therapeutic results are reported or even suggested. Blisterings and cuppings, vascular sedatives and pilocarpine sweatings, iodides, quinine and salicylates have all been thoroughly exploited with no positive benefit. If only there were a method of applying modern surgical decompression to the labyrinth capsule there might be hope of saving the delicate end organs. Spinal puncture should be tried even though there be no direct communication with the endolymph. Lower tension in the subarachnoid may drain the perilymph and thereby reduce intracapsular tension. In fact there is a possibility that the hypertension is confined to the perilymph.

The important thing is to give to epidemic parotitis a more dignified place in otology and pathology. It is worthy of scientific study in epidemic groups particularly and if hypertension is the destructive agency a method of direct or indirect decompression may be discovered.

Epidemic parotitis is caused by a diplococcus which excites a violent inflammation without tendency to suppuration.

The infectious bacilli are thoroughly disseminated throughout the system.

The variable character of clinical symptoms in point of severity and different organs involved, and the wide range of susceptible age tend to obscure the diagnosis and not rarely the primary infection is overlooked. It is a fact that labyrinthitis and partial or absolute deafness may be clinically the only positive manifestation of the disease, and the same is true of orchitis.

Hypoacusis of obscure origin can occasionally be traced to mumps.

The fact that the most serious sequel of mumps is destruction of the function of the labyrinth suggests the explanation based on the anatomical peculiarities of this organ. This is delicate tissue formation within an unyielding bony capsule.

Hypertension possibly confined to the perilymph is probably the destructive factor in nonsuppurative labyrinthitis association with parotitis epidemica.

BIBLIOGRAPHY.

1. Urbanschitsch. Lehrbuch der Ohrenheilkunde, 1910.
2. Haug. Arch. int., de laryngol. 1907, XXIII, 720.
3. O. Mauthner. Wein Med. Wochenschrift, 1911, LXI, p. 2089.
4. Chapelier. Arch. gen de med. 1905, 88, 2881.
5. Jacobson. Lehrbuch der Ohrenheilkunde, 1898.
6. Ostman. Lehrbuch der Ohrenheilkunde, 1909.
7. Boot. Jour. Am. Med. Assn. Dec. 5, 1908.
8. Herb, Isabella C. Arch. Int. Med. Sep., 1909.
9. H. Haike. Zeitschrift fur Kinderheilkunde, 1911, Bd II, p. 497.

FOCAL INFECTION IN THE ETIOLOGY OF LABYRINTH DISEASE.

By GEORGE E. SHAMBAUGH, M. D., Chicago, Ill.

In recent years a great deal has been done to clear up the various problems in the physiology of the internal ear. With this has come a better understanding of the symptoms arising from disturbance in function when the labyrinth is invaded by disease. We experience, as a rule, little difficulty in diagnosing cases of labyrinth disturbance and of reaching a more or less satisfactory conclusion regarding the probable nature of the pathological changes which have taken place. The diseases of the labyrinth which we readily recognize include labyrinth involvement from suppurative disease of the middle ear or from meningitis, injury to the labyrinth either through a fracture of the base of the skull or when operating on the middle ear, syphilis of the internal ear, otosclerosis with labyrinth involvement, degeneration in the labyrinth following long standing obstructive middle ear deafness, the degeneration in the labyrinth occasionally, following the infectious fevers, as well as occupational deafness.

There remains a group of cases in which the labyrinth disease occurs in patients who have had no middle ear trouble and no general disease to account for the labyrinth involvement. In some of these cases the symptoms arise from disturbance limited to the cochlea; in others the symptoms are from involvement of the vestibular apparatus alone, while in many of the cases there is a disturbance of the whole labyrinth. The symptoms arising from the involvement of the cochlea are nerve deafness and usually tinnitus aurium. The deafness may be very insidious in its progress, in which case the tinnitus may be very slight or even quite absent. In many of the cases the progress of the deafness takes place chiefly by acute exacerbations, which occur as distinct apoplectiform attacks, and in these

cases the tinnitus is always more marked immediately following each separate attack. In a few cases the first attack destroys permanently the function of hearing. The accompanying tinnitus aurium in such cases gradually diminishes but often lasts for a period of years before entirely disappearing.

The symptoms arising from disturbance of the vestibular part of the labyrinth are those of disturbed equilibrium, the result of the unbalance occasioned by the partial or complete suppression of tonus from one labyrinth. This disturbance is always temporary and occurs as the sequel of an acute exacerbation of the vestibular involvement. In cases where the vestibular involvement is very insidious in its development the symptoms of disturbed equilibrium may be entirely absent.

The clinical picture from this group of labyrinth cases differs, therefore, very widely, depending upon whether the trouble is insidious in its development or whether it is punctuated by distinct apoplectiform attacks, and upon whether the disease involves both parts of the labyrinth or is restricted to either the cochlea or the vestibule. When the disease is associated with acute exacerbations involving the whole labyrinth it gives rise to the group of symptoms which are commonly known as the Mennier-symptom-complex, that is, deafness, tinnitus and vertigo. When the disease involves both parts of the labyrinth but progresses without acute exacerbation, the only symptoms are deafness and tinnitus.

It is in regard to the etiology of this group of labyrinth cases that I wish to call your attention. Mennier was the first to associate the attacks of vertigo, when combined with deafness and tinnitus, with disease of the internal ear. He believed that the disturbance was the result of hemorrhage in the labyrinth. That a hemorrhage into the labyrinth may produce this group of symptoms no one now will deny, but there are no grounds for believing that this is the actual pathological change which takes place in these cases.

Some of these cases are called essential neuritis, others are designated as chronic progressive labyrinthine deafness,

but there is nothing in either of these terms to suggest the cause of the changes going on in the internal ear. When we seek to find analogies for these internal ear degenerations among diseased conditions found elsewhere in the body, we recognize at once the similarity between the phenomena observed in these labyrinth cases and the chronic degenerations which occur as the result of focal infection. Recent investigations have shown that chronic arthritis and neuritis, chronic cardio-vascular degenerations and chronic nephritis, are the result frequently of chronic latent foci of infection, the presence of which is often not suspected by the patient since they often produce very slight or no local symptoms.

In all of these systematic diseases there is the chronic progressive character, punctuated, as a rule, from time to time by acute exacerbations, which are accounted for by a fresh shower of bacteria discharged from time to time into the circulation from the infected focus. These bacterial emboli lodge now in one part, now in another, through some as yet unknown predilection. Sometimes only one part, as a single joint, is involved; in other cases several structures are simultaneously affected.

To account for the phenomena observed in many of these cases of internal ear disease as the result of focal infection, we have only to assume that the endings of the eighth nerve may be the structure peculiarly susceptible to the bacteria liberated from the infected focus. Each fresh shower of bacteria liberated into the circulation produces a sudden suppression of function either in the cochlear or vestibular nerve endings, separately or in both simultaneously. With each attack the patient experiences a defect in the hearing associated with tinnitus aurium, when the cochlea alone is involved, or there is a disturbance of equilibrium with vertigo resulting from the one-sided suppression of labyrinth tonus when the vestibular apparatus is involved. When both the cochlear and vestibular endings are simultaneously affected we have as a result the complete picture of the Menniersymptom-complex: that is, deafness and tinnitus with vertigo.

A single attack may occasionally be severe enough to produce a total permanent suppression of function in the affected labyrinth. Here in addition to the total deafness in the affected ear there will be a severe tinnitus, which diminishing gradually after the attack, may still require years before it entirely disappears. The vertigo occasioned by the sudden complete unilateral suppression of labyrinth tonus will be very marked but will also gradually disappear, rarely lasting more than a few weeks or months. In such cases, where the single attack causes a complete destruction of function in the affected labyrinth, there will, of course, be no subsequent attacks, unless the other ear should later become involved.

In most of the cases there is but a partial suppression of function resulting from a single attack. Here the deafness, as well as the tinnitus, are most marked immediately following the attack, but neither disappears entirely, as a rule, even after a long period. The vertigo usually disappears rather promptly after a few days or weeks. In these cases of partial suppression of labyrinth function there will very likely be subsequent attacks, provided the focus of infection persists. In some cases the attacks with the Mennier-symptom-complex persist in recurring at irregular intervals over several years, associated always with an increasing loss of function. The attacks may cease only after the function of the affected labyrinth has been completely destroyed.

With this conception that the internal ear may be the target for a systemic infection of focal origin, we have at once a plausible explanation not alone for the chronic progressive character of the degeneration going on in the labyrinth, but also for the apoplectiform attacks, with which these degenerative changes are so prone to be punctuated.

The possibility that some of the labyrinth cases of unknown origin may be caused by focal infection cannot be denied. It behooves us therefore, when examining these labyrinth cases of obscure origin, to make a careful search

for foci of infection, just as we have learned to do in cases of rheumatism and allied conditions.

The focus for these systemic infections is usually found in the faucial tonsils or in abscesses around the teeth. When the tonsils contain the foci producing the systemic infection there is very often a history of recurring attacks of acute tonsillitis. In some of the cases the patient will admit having recurring attacks of "sore throat" but denies ever having had attacks of tonsillitis. As a matter of fact the recurring attacks of sore throat are usually mild attacks of tonsillitis, as can be readily determined by examining the pharynx during one of the attacks. It is, however, not uncommon to obtain no history of sore throat, and yet a careful examination will disclose unmistakable evidence in the faucial tonsil of chronic infection. In some cases this evidence is the presence of pus, which can be expressed from the tonsil. In others the congested appearance of the tonsil indicates clearly the presence of an infection. Such tonsils may be enlarged but often they are distinctly shrunken and smaller even than the normal tonsil.

When the focus of infection is located about the teeth it not infrequently requires the use of the radiogram to discover the abscess.

Since this relation between foci of infection and disease of the internal ear suggested itself to me several years ago, I have had the opportunity of examining a large number of these cases of internal ear disease of obscure origin and have already collected a number where the presence of such chronic foci of infection could be demonstrated, which our experience has shown to be capable of producing systemic infection. I will give briefly the history of three typical cases.

Case I. Mr. S., age 44, seen in April, 1912, because of a sore throat. Faucial tonsils had been operated on five years before. Large stubs had been left on both sides. Tonsil on left side congested. On pressure pus could be expressed. June, 1914, he consulted me because of a soreness in the left tonsil which had persisted after an acute tonsillitis a few

weeks before. Pus expressed from the left tonsil showed pure culture of streptococcus viridens. Patient had an acute swollen joint in the forefinger of right hand. Ten days later had an attack of vertigo with nausea, which confined him to the house for several days; slight tinnitus aurium, but no defect in the hearing. Membrana tympani normal and no evidence of middle ear disease. Patient seen again in October, 1914. Had developed rheumatic soreness in several joints, including hands, neck and knees. Pus still found in left tonsil. Tonsil stubs enucleated Nov. 7th. Pure culture streptococcus viridens grown from the tonsil. Following the operation the rheumatism disappeared for two months, when he began to have recurrence. The Roentgenogram disclosed a tooth abscess. The tooth was removed. Pus from the abscess gave pure culture of the streptococcus viridens. The rheumatism disappeared promptly after the removal of the focus of infection under the tooth.

Case II. Mrs. L., age 53, consulted me October, 1911, because of defective hearing. The trouble had developed suddenly 3 years previous in association with an acute peritonsillar abscess on the right side. She had experienced a sudden attack of deafness and tinnitus in the right ear, associated with vertigo and sensation of objects moving before the eyes. Examination: drum membranes normal and the functional tests disclosed complete deafness in the right ear with slight loss of hearing in the left.

Case III. Mrs. M., age 68, consulted me May, 1914, because of a very severe attack of the Mennier-Symptom-Complex, during which the vomiting had lasted for 24 hours. History of similar but lighter attacks for several years. Has been somewhat hard of hearing for a number of years. Has had chronic articular and muscular rheumatism for several years. History of recurring sore throat. Tonsils were small but presented the congested appearance characteristic of tonsils the seat of chronic infection. Pus was expressed from the right tonsil. Both ears hard of hearing; whisper not heard in left, heard in right about 2 feet; deafness of the labyrinth type.

Discussion.

Dr. Max A. Goldstein, St. Louis, in connection with Dr. Hubbard's paper, emphasized the point that it is difficult, in cases of mumps where the toxic neuritis has taken place in the ear, to differentiate from ordinary labyrinthitis in which the toxic process follows along the nerves. It was a mooted question, in cases of labyrinthitis associated with mumps, whether the labyrinthitis was the result of the mumps infection or whether it was a deep-seated pathological problem.

Dr. B. Alexander Randall, Philadelphia, suggested that in mumps cases with aural complications one must be very thorough in one's investigation in order to escape mistakes. He cited a case in point. A well-developed girl of fifteen came with nearly total nerve deafness ascribed to mumps. She was accompanied by a cousin of the same age, so that a satisfactory history could not be obtained. The girl had beautiful teeth. He was about to accept her statement about the deafness being due to mumps, when he noticed that her eyes had faint circum-corneal injection and the ophthalmoscope showed interstitial keratitis. He then discovered that her teeth were artificial. He could obtain no very definite family history at subsequent visits, as to miscarriages, or other points that would tend to confirm his suspicion that she had hereditary syphilis, but her prompt response to antisyphilitic treatment convinced him that both the eye and ear condition were syphilitic. He raised the point, in connection with focal infections, that while the members of the Society were on the lookout for microorganisms of a vegetable nature, they had not given as much consideration to the ameba as the subject deserved. Pyorrhea alveolaris had come to be so clearly associated with the ambea that it raised the question as to what part this organism might play in cases such as those reported by Dr. Shambaugh. Examination of pus not only from the ear but elsewhere should involve examination for the ameba. A mere smear, hardened in alcoholic

solution of bichlorid, will fix the ameba in a way that makes the diagnosis fairly easy; and it has been thus found in carpus and even in otitic brain lesions.

Dr. Hubbard, in closing the discussion, said he excluded the toxic neuritis theory as the cause of deafness in mumps because of the suddenness of the onset. Hypertension is the cause of the destruction of the delicate nerve function. This is probably confined to the perilympth of the labyrinth. This might be determined clinically by lumbar puncture during the incipiency of labyrinthine symptoms.

THE DIFFERENTIAL DIAGNOSIS OF LESIONS OF THE LABYRINTH AND CEREBELLUM.

By J. GORDON WILSON, M. A., M. B., Chicago, Ill.

In this review I shall confine my remarks to three symptoms common to lesions in the labyrinth and in the cerebellum, viz: nystagmus, vertigo and ataxia. With the time at my disposal it is impossible to discuss adequately these three symptoms in their various modifications by extension of the disease or its effects to surrounding structures, so I shall chiefly consider lesions which are strictly and definitely labyrinthine and lesions which are strictly and definitely cerebellar. I hope thus to be able to emphasize the points common to each and to note essential differences, if any, and thus arrive at the intelligent understanding essential to diagnosis.

When one talks of a lesion definitely labyrinthine and definitely cerebellar, one immediately thinks of experimental lesions. As Dejerine has pointed out (Dejerine, Semiologie des affections du Systeme Nerveux, Paris, 1914, p. 436), clinical lesions are not often confined, so far as their effects go, wholly to the cerebellum. An abscess of the cerebellum is accompanied by the production of toxins, which act, through the medium of the circulation, upon neighboring or even upon remote structures; a new growth in the cerebellum brings about changes in the general intracranial pressure, resulting in changes in the cerebral circulation and usually, also, in an increased pressure of the cerebrospinal fluid. (Frazier, Journal of the American Medical Association, 1915, lxiv, p. 1121.) The increased pressure of cerebrospinal fluid will in itself often lead to vertigo, quite apart from the destructive effect of the neoplasm, and the physical pressure of the tumor upon the medulla oblongata and the floor of the midbrain may give rise to other phenomena not directly related to the structures involved in the lesion.

While the clinical picture may be necessary for diagnosis, the experimental picture must not be neglected in drawing conclusions with reference to normal function, and disturbance of function must be the basis for a rational diagnosis.

While much of what is presented here is based on experimentation on animals done by Dr. Pike and myself, it has received sufficient verification in man, both in cases we have observed and cases recorded by others,—often, as in the case of cerebellar nystagmus, without a full appreciation of the significance of what is recorded (e. g., Stewart and Holmes, Brain, 1904, vol. 27, p. 529).

When we consider how close anatomically and physiologically the labyrinth is to the cerebellum, and when we recall that many of our labyrinthine cases when they come under observation have affected the posterior cranial fossa with more or less involvement of the cerebellum, and that so many cerebellar lesions are complicated by pressure on the vestibular nerve, it is little wonder that the most careful of observations leave us often hopelessly involved. It is for this reason that I feel tempted to try to outline the essential differences of labyrinthine and of cerebellar lesions.

It will be well here to attempt to define the physiological function of the labyrinth and of the cerebellum. This appears necessary because so much confusion has arisen between the primary disturbance and the remote effect associated with lesions in these organs.

The primary function of the labyrinth is to give information of the change of position of the head in space (Wilson and Pike, Some Considerations on the Physiology of the Otic Labyrinth, Arch. for Internal Medicine, 1914, xiv, p. 911—920). Its end organ signals all movements of the head or more correctly, all acceleration of movement. Rapid changes of aspect of the head in space demand prompt and rapid motor reactions to preserve equilibrium or to break a fall. A moment's thought will lead us to see the wide reaching range of its reflex associations. Thus it must be in touch with all the mechanism which tends to readjust the body and be able to call into play compensatory movements to

maintain equilibrium. There is no other sense organ in the body which will give this information quite so efficiently as the labyrinth, and hence, there is always a certain amount of permanent deficiency of motor response after total loss of the otic labyrinths.

As a result of this we find (1) that the labyrinth is in such direct touch with the eyes that each movement of the head may have, when necessary, such compensatory movements of the eyes as are necessary to maintain the normal relation of the eyes to the horizontal. (Sherrington, the Integrative Action of the Nervous System, New York, 1906, p. 337). This as we have shown (Wilson and Pike, The Effects of Stimulation and Extirpation of the Labyrinth of the Ear and its Relation to the Motor System, Royal Soc., Phil. Trans. 1912 vol. 203, p. 127-160) is a direct reflex response independent of the cerebellum with the probable object of ensuring that retinal images shall not be disturbed by each movement of the head. (2) That the labyrinth is intimately connected with the body musculature so as to prevent loss of balance. When the usual limit of stimulation of the labyrinth is over stepped there are developed among other symptoms, nystagmus, and a fall to the side of the lesion, or to the side of the less intense stimulation.

The fundamental action of the cerebellum is not so easily expressed nor are investigators agreed in regard to its full significance. Sherrington would place it as the ganglion of the proprio-ceptors, that is, those end organs or receptors which are buried in the body and adapted to react to stimuli developed in their neighborhood in the organism itself (Sherrington, loc. cit. p. 348 and 130). It thus becomes a great co-ordinating centre for the proprio-ceptors and its destruction renders difficult or impossible the associating of movements directed to the same end.

Babinski says that normally the cerebellum exercises a control action upon voluntary movements. Mills thus expresses his conception "the fundamental symptom in Cerebellar disease is Asynergy—defining Synergy as that power or faculty by which movements more or less complex, but

functionally definite, are associated in a special act or acts." He quotes with approval Ferrier's idea of the cerebellar activity "Every form of active muscular exertion necessitates the simultaneous co-operation of an immense assemblage of synergic movements throughout the body to secure steadiness and maintain general equilibrium." These ideas are fundamentally alike—so we will not be far from the current views when we say that the cerebellum is of importance, to correlate such unconscious sensations from muscles and joints as assure the continuity and proper degree of motor activity in all our movements. In our opinion, Luciani's objections to considering the cerebellum as the great central organ for the maintenance of equilibrium have not been satisfactorily met. (Luciani, Human Physiology, London, 1915, v. 3, p. 448).

Mills classes as special symptomatic manifestations of asynergy: dysmetry (the lack of proper proportion or measure of movement in time and space), adiadokokinesia, and tremor. As symptoms resulting from asynergic efforts: asthenia (lack of motor power), atonia, and ataxia. Since the loss of strength is never complete, Sherrington (Sherrington, Schaefer's Textbook of Physiology, 1900, ii, p. 903) characterizes these conditions as parasthenia and paratonia.

One sharp distinction between cerebellar function and labyrinthine function lies in the fact that the cerebellum is an afferent station for the proprioceptive impulses and the labyrinth is not. Impulses simply originate in the labyrinth. The cerebellum lies in one efferent path from the cerebrum to the cord; the labyrinth does not. The labyrinth is a peripheral sense organ.

Nystagmus—Labyrinthine.

Nystagmus occurring as a result of a labyrinthine lesion consists of two phases, a slow lateral deviation, labyrinthine in origin, followed by a quick return movement, cerebral in origin. This idea, expressed by us in former papers, has been confirmed by all our subsequent experiments, still unpublished. On the basis of these experiments, we feel

safe in saying that the impulses leading to the quick movement in labyrinthine nystagmus comes from the cerebral hemisphere on the side to which the slow deviation of the eyes is directed. The quick movement fails when the cerebral hemisphere to the side of the slow deviation of the eyes is extirpated. These movements, are synchronous in both eyes and vary definitely in direction with the labyrinth which is affected. In lesions produced experimentally or due to pathological processes in the labyrinth, the movements are primarily horizontal, and it is the horizontal movement that is of importance. The rotatory movement apparent at times, so far as my present knowledge goes, does not appear to have much diagnostic value, and insistence on it appears to me to confuse the issue. When observed, this rotatory movement is designated according to the direction of the quick movement at the upper end of the vertical iris-meridian. In nystagmus, after experimental lesions, the rotatory movements are of secondary importance and are most noticeable very early and again as the nystagmus is passing away.

In experimental work, the deviation is always to the side of the lesion. I have never seen a case in which the deviation was to the side opposite the lesion so long as one labyrinth remained intact. At first, there is at times a slight vertical action, especially on the side of the lesion, the eye deviating slowly down, but even here it tends to the side of the lesion. This stage, however, passes in a few hours. As has been noted elsewhere (Wilson and Pike, The Effects of Stimulation and Extirpation of the Labyrinth of the Ear and Their Relation to the Motor System, Royal Society, Philosophical Transactions, v. 203, p. 127-160), in the very early stages the deviation can be made to go to the side opposite the lesion by mechanical pressure.

An important question here arises as to whether we ever have a labyrinthine lesion with deviation away from the side of the lesion; that is, with the quick phase to the side of the lesion. Theoretically, it is easy to reply that in the early stages of labyrinthine disease we have an irritation or stim-

ulation of the ampullary nerve endings which will produce such a deviation and nystagmus. All the observations we have made leave no doubt in our minds that the normal stimulus for the end organ is pressure. But the pressure which arises in the course of a labyrinthine effusion is diffuse and different from the pressure arising from the definitely directed physiological pressure of the fluids in the canals. The pathological effusion in such a small confined cavity rapidly becomes general, if it be not so from the first, and results in a depression of activity. We have no evidence that generalized pressure will act on the nerve terminals to cause an irritative lesion.

In a serous labyrinthitis the effusion may be into the perilymph or the endolymph. On general principles one would say that the evidence at present shows that in many cases the increase is in the perilymph especially in cases which result in complete recovery. In serous labyrinthitis the deviation is to the side of the lesion, the quick phase is to the sound side. In the cases recorded when the slow deviation occurs to the sound side during the course of an acute or chronic otitis media or following a mastoid operation but without the presence of a fistula, in spite of the opinion of Alexander that this indicates a stage of irritation (Ruttin, Klinik der Seröson u eitrigen Labyrinth, Entzundungen, Wein, 1912, p. 83) it appears to me we are dealing with an irritation outside of labyrinth. In the cases of nystagmus to the diseased side which I have observed occurring either during the course of an otitis media or after a mastoid operation the deviation away from the side of the lesion had such additional symptoms (pain over the region of the fifth nerve, or paresis of the sixth or seventh) as pointed to some distinct involvement of the petrous temporal.

There is an interesting table given by Ruttin (loc. cit. p. 39) of the direction of the quick phase of nystagmus. He there tabulates two varieties of labyrinthine lesions in which *on looking to the right or to the left* the general rule of slow deviation to the side of the lesion is not followed. These are (1) circumscribed labyrinthitis in which the quick phase

is directed to the diseased side or to the sound side or to either side—to the right on looking to the right, to the left on looking to the left, and (2) diffuse suppurative latent labyrinthitis in which also the quick phase is directed to either side on looking to that side.

In circumscribed labyrinthitis the irregularity of the nystagmus is associated with a membranous labyrinth more or less intact but with a fistulous opening in the bone. Ruttin (loc. cit. p. 35) thus exemplifies it "A patient with chronic middle ear discharge has complained for some time of vertigo and perhaps also of tinnitus. On examination there is more or less deficiency of hearing but not complete loss, the fistula test is positive, the caloric reaction is present, spontaneous nystagmus is present, and often toward both sides, that is, on moving eyes to right or left voluntarily." In the diffuse suppurative latent labyrinthitis we have a mechanical irritation of the nerve often associated with caries of the petrous temporal.

The explanation in these chronic cases of this irregularity in the labyrinthine reaction on looking to the side is not very obvious, but we suggest that it is due to irritation of an inflamed vestibular nerve. A suggestion of Luciani (Luciani, Physiologie des Menschen, Jena, 1907, Bd. iii, p. 309, and Luciani, Human Physiology, London, 1915, p. 204) is perhaps applicable here. Claude Bernard in his experiments on the nerves of recurrent sensibility, was unable to get his most pronounced sensory effects on stimulation of the peripheral portion of the ventral nerve roots immediately after the nerve roots were exposed in the spinal canal; the sensory effects were noticeable only when stimulation was done at least one hour after the nerve roots were exposed. Bernard attributed this delay to the time required for the recovery of the nerve trunks from the shock resulting from division of the ventral roots. Luciani is not inclined to credit this hypothesis of shock, but inclines to the belief that the nerve trunks become more sensitive to stimulation in the course of a few hours because of the development of the inflammatory reaction in the exposed nerve roots. It is common

knowledge that inflammatory processes in the joints or elsewhere often render the nerve endings of affected regions more sensitive to mechanical pressure, but it is equally well known that specialized nerve endings suffer early from inflammatory processes. While it is possible that the vestibular nerve trunks might be rendered more susceptible to stimulation by the development of the inflammatory reaction along their course, inflammation within the ampullae would be more likely so to injure the cristae acusticae as to alter the normal threshold for pressure from the canals as a stimulating agent. Thus it may be that the variations in blood pressure in a localized area, e. g. in a granular mass projecting through a fistula into a canal—is an efficient cause.

We believe that the nystagmus in both directions, namely, in looking to the right the quick phase to the right, the slow to the left, and in looking to the left the quick phase to the left and the slow to the right, can be best explained by our hypothesis of two distinct sets of nerve fibres in the vestibular nerve for reasons already given. This hypothesis, as we have already explained, is based essentially on Lee's findings that the differences in direction of movement of the eyes varies with the intensity of mechanical stimulation of the vestibular nerve endings, and our own observations on the results of stimulation when but one labyrinth is present.

According to this hypothesis, the vestibular nerve contains two groups of nerve fibres functionally distinct:

(a) A numerous group of fibres excited by a strong stimulus turning the eyes to the opposite side.

(b) A less numerous group of fibres more easily excited and hence affected by a weaker stimulus turning eyes to the same side.

In an inflammation of the left vestibular nerve, both sets of fibres are affected and stimulated. On looking to the left, a cerebral act, the left internal rectus and the right external rectus are relaxed, so far as cerebral impulses are concerned. The labyrinthine impulses are, however, still coming in to these muscles since there is no reason to suppose

that cerebral action has any effect upon the inflammation along the course of the vestibular nerve.

Sherrington (Sherrington, Proceedings of the Royal Society, London, 1893, vol. 53, p. 412,) has shown that electrical stimulation of the cortical area for movement of the eye muscles will, as an example, bring about contraction of the left external rectus and the relaxation of the left internal rectus, and that the mechanism for the inhibition of the antagonistic muscles is subcortical. From this it will be seen that the suppression of the quick phase in labyrinthine nystagmus on voluntarily turning the eyes in the direction of the slow phase is associated with the relaxation of the opposing muscles, whose tension we believe, is the primary cause of the quick movement. In any case of cortical relaxation or inhibition of a muscle, the relaxation is never complete, and the muscle is never slack. A skeletal muscle always gapes when cut transversely. In the lesions under consideration (say of left labyrinth), in looking to the left the stimulus coming to these relaxed fibers from the stimulation of one set of fibers from the left labyrinth causes a contraction of these muscle fibers and a slow movement to the right. Similarly in looking to the right, the left external rectus and right internal rectus are relaxed, so far as cerebral impulses are concerned, and the stimulus from the other set of left labyrinthine fibers causes a slow movement to the left. There thus results a double nystagmus on directing the eyes to the right or the left.

I have so far only considered pressure on the terminal organ in the labyrinth. In lesions involving the vestibular nerve intracranially—causing pressure on or irritation of the nerve—there may be a deviation away from the side of the lesion. Many of the so-called deviations of nystagmus are cerebellar or jointly cerebellar and labyrinthine and as such do not come within the scope of this paper.

Nystagmus-Cerebellar.

We have much to learn concerning cerebellar nystagmus. For instance we do not know sufficiently accurately the localities in which lesions may occur yet be associated with

no nystagmus; we have not sufficient data to differentiate between an irritative and a destructive lesion. There are numerous experiments which show that cortical irritation of the cerebellum with the electrical current produces no effect so long as care is taken to limit the excitation to the cortex. Luciani, Risien, Russell and Horsley have shown that stimulation of the dentate nucleus produces deviation of the eyes to the side of the nucleus stimulated.

The various articles descriptive of cerebellar nystagmus appear to me to be lacking in that clearness and definiteness which we are arriving at in labyrinthine nystagmus. Here one is trammelled by observations made on cases which are not definitely cerebellar, as well as by lack of appreciation of some of its characteristics. I do not propose to weary you with more than one quotation to exemplify this, and this I take from the school which has done much to advance the clinical applications of the physiological facts, although, it must be confessed, with some violence to these facts. Neumann (Neumann, Otitic Cerebellar Abscess, London, 1909, p. 18), in his work on cerebellar abscess, says:

"The focal symptoms of disease of the cerebellum are due on the one hand to lesion of Deiter's nucleus and the afferent and efferent tracts, and on the other hand to lesion of the secondary tracts, which belong to the same side of the body, and stand in relation to the co-ordination of muscular sense. The lesion of Deiter's nucleus produces vestibular nystagmus, vertigo, and vestibular ataxia." "As regards nystagmus, this is always of a rhythmical character, and shows on the whole the characteristics of a vestibular nystagmus thus according with the description given by Bárány. The differentiation of vestibular nystagmus of cerebellar origin from that originating peripherally in the semicircular canals is made on the one hand by careful functional testing of the vestibular apparatus, similar to that carried out by Bárány in our clinic; whilst, on the other hand, the course of the disease and the accompanying symptoms assist in the diagnosis."

If the description here given sums up the current opinion of cerebellar nystagmus I cannot agree with it. Exception

has to be taken to each sentence. If the first sentence means anything it means that cerebellar nystagmus is due to lesions of Deiter's nucleus and appears based on the erroneous idea that the labyrinthine nystagmus is dependent upon an anatomical course through the cerebellum. This we have shown is erroneous. So far as the second is concerned of course a lesion of Deiter's nucleus will produce vestibular nystagmus because as has been shown it lies on the central path between the afferents from the ear and the efferents to the eye. We do not believe that cerebellar nystagmus shows on the whole the characteristics of labyrinthine nystagmus. Unfortunately this teaching has formed the basis for most of our text book articles. It lacks sound anatomical and physiological basis and is not in keeping with experimental results. More than that it is not in keeping with exact clinical observations.

Experimental work early impressed on us that if there be one thing characteristic of cerebellar nystagmus it is that it is not comparable to labyrinthine nystagmus. It differs from it:

(a) in uniformity of direction; and (b) in character.

Some years ago we stated that cerebellar nystagmus was essentially ataxic and we have seen no reason to alter that opinion. The great and distinct feature is its character—its oscillatory nature approaching towards an ataxia of the eye muscles. With the animal at rest it is lessened or may disappear. It is increased by fixation—especially at the beginning and during the intensity of the fixation. In other words it is to be regarded as an asynergia. Behind glasses when the patient ceases to focus it tends to become less marked, and here again differs from labyrinthine. This is in keeping with what has been said above in regard to the known function of the cerebellum. A lesion here will produce an incoordination of movement; and movements of the eye muscles offer no exception.

In direction the slow deviation may be away from the side of the lesion though it may be to the sound side depending on whether we are dealing with an irritative or destructive

lesion. But it tends to be irregular in the plane of its direction with the head at rest.

Vertigo.

Vertigo as we have stated elsewhere (Wilson and Pike, Journal of the American Medical Association, 1915, v. 64, p. 561-564) is the confusion resulting from the coming into consciousness of afferent impulses concerned with orientation in space which ordinarily are associated (integrated) but now for some reason have become dissociated. I will not concern myself here with the varieties of sensations which are embraced under the term vertigo, the majority of which are too indefinite to be of value in diagnosis. I will only refer to vertigo associated with the sensation of rotation. It is well to emphasize that in vertigo we are dealing with a subjective sensation and as has been said elsewhere (Wilson and Pike, loc. cit. p. 563) "One notes how difficult it is to get a distinct history of subjective sensations even from intelligent patients, and further how much less reliable are the subjective sensations of apparent movement of external objects."

The law applicable to apparent rotation following stimulation or destruction of the labyrinth is that apparent rotation of one's self is always in the direction of the rapid phase of nystagmus. There is not the same unanimity in regard to the apparent rotation of objects, "In post-rotary visual dizziness Bárány sees the visual field oscillate from side to side in both directions; whereas Mach, Breuer, Delage and most other observers see it whirl contrariwise to the preceding actual rotation; and Helmholtz saw it whirl sometimes with and sometimes contrary to, the direction of the preceding rotation. (Holt, On Ocular Nystagmus and the Localization of Sensory Data during Dizziness, Psychological Review, Nov. 1909, vol. 16, p. 388). My own observations confirm the findings of Holt that as a rule the post-rotary apparent rotation of objects is contrariwise to the preceding actual rotation, that is, in the direction of the quick phase of nystagmus. If no nystagmus be present the

apparent rotation of self points to the side of the lesion. (Wilson and Pike, loc. cit. p. 563).

Less uniformity exists in cerebellar cases. On this point careful observations were made on a series of 40 cases of tumors involving the cerebellum by Stewart and Holmes (Brain, 1904, v. 27, p. 525). The direction of the subjective movements were so constant that they were led to regard this as of serviceable diagnostic value. They found:

"(1) In both intra- and extracerebellar tumors the sense of displacement of external objects in front of patients is from the side of the lesion to the opposite side. Thus if the tumor be on the left side, the apparent movement of objects is from left to right.

(2) In intracerebellar tumors the subjective rotation of one's self is always from the side of the lesion to the healthy side, that is, in the same direction as the apparent movement of external objects.

(3) In extracerebellar tumors the subjective rotation of one's self is always from the healthy side towards the side of the lesion, that is, in the opposite direction to that of the displacement of external objects."

It will be noted that the apparent displacement of external objects is similar to that generally occurring in labyrinthine lesions, and that the apparent rotation of one's self varies with the position of the tumor. The relationships in intracerebellar tumors are the same as in labyrinthine lesions. The diagnostic importance of the direction of subjective movements in cerebellar lesions has been much questioned, for instance, by Oppenheim (Lewandowsky, Handbuch der Neurologie, Berlin, 1910, p. 843); but even if not generally applicable, they offer suggestions of value to aid in the diagnosis.

Ataxia and Dis-orientation.

When we talk of ataxia, we mean inco-ordination of movement—a disturbance in the particular organ itself. When we talk of disturbance of orientation, we mean that the organism has a special mechanism which adjusts the normal

relationship of the body and its movements to its position or change of position in space. The organism has numerous peripheral organs which signal in any change of position in a particular part—the ear, eye, muscles, joints, etc. As the head is the most important segment of the body, so without doubt the labyrinth and the eyes are the most important receptive organs for the sensations necessary for orientation. This is seen in the difficulties in walking of our labyrinthless dogs with the eyes bandaged and the difficulties that deaf mutes and labyrinthineless dogs have in swimming. In the latter case, the absence of contact with solid bodies and in a medium in which appreciable differences of external pressure are excluded, the labyrinth is the only agency capable under all conditions of giving adequate information of the change of position of the head in space. The action of the eyes is apparently less general and slower, but this will be discussed more fully in a later paper.

All these inflowing messages have to be correlated to produce a harmonious action of the whole organism—and probably the cerebellum is one organ which participates in this. This correlation no doubt involves the afferent fibers which the cerebellum receives from the vestibular nerve, and its very close relationship with the vestibular nuclei in the pons and medulla. While it is probable that the cerebellum participates in the integration of the impulses from the labyrinth with others from the periphery into the higher uniform function of maintaining a given orientation in space, yet certainly the cerebellum is not the sole central organ for orientation. In passing, it may be noted that it is this disturbance with the inflowing impulses from the periphery that is the scientific basis of our attempts to localize particular cerebellar areas.

In both cerebellar and labyrinthine lesions there is a deviation in walking to one side—to the side of lesion, if it is unilateral. With the eyes open, the patient corrects this; with the eyes shut, he may correct it, but usually, and especially in cerebellar cases, corrects too much and then deviates to the sound side. This deviation has nothing to do with

ataxia. It is a disturbance of orientation in space, is involuntary, and appears in its most pronounced form in animals. The sensation of such involuntary movements which is common in cerebellar disease must correspond to objective involuntary movements.

When we consider the disturbances of orientation and the ataxia common to cerebellar and vestibular lesions, we find as the most obvious symptom that the patient stands with the feet spread wide apart, that there is inability to stand on one foot, that the gait is uncertain, and that the body tends to turn to the right or the left. A discussion of the variations of muscular strength or of muscle fatigue does not come within the scope of this paper.

Labyrinthine lesions differ from cerebellar in (1) the existence of Romberg's sign in labyrinthine lesions, (2) variations in the attitude of the head influence the lack of equilibrium of the body in labyrinthine and not in cerebellar lesions, (3) an affection of the labyrinth does not definitely involve those movements of isolated parts which result in dysmetria, (4) in labyrinthine disease movements of rotation or disorientation are not so readily perceived; labyrinthine nystagmus and rotation vertigo are lost; electrical and caloric tests are modified or negative.

(1) Romberg's sign. The disturbances of equilibrium during walking and standing are increased in labyrinthine disease by shutting the eyes. Suppression of visual control does not have the same effect in cerebellar lesions. Sight is of less use to the cerebellar patient in maintaining the erect position although probably of some use and there are said to be (Lewandowsky, loc. cit. p. 842) cerebellar cases where Romberg's sign is positive, but as Dejerine insists, they are rare. Romberg's sign is rare in destructive lesions of the cerebellum, as atrophy or sclerosis; rather frequent in abscesses, tumors, or in lesions which affect other structures in their vicinity, and one may seriously question whether in the cases of which Bárány speaks the cerebellum alone is involved, either directly or indirectly. In labyrinthine disease standing on one foot is more difficult especially on the foot on the

side of the lesion. This, however, is of no value if the patient has a peripheral sensory disturbance.

(2) Relation of the attitude of the head to the disturbances.

(a) Goltz and Ewald showed many years ago that the pigeon deprived of its semicircular canals is not able to maintain its balance if the position of the sustaining base is changed. This experiment has been repeated by many workers in other animals, and on it is based Stein's goniometric test. This consists in placing a patient with bandaged eyes on an inclined plane the slope of which may be varied. It is found that in labyrinthine cases the patient does not perceive, or perceives but slowly, the changing inclination of the plane and lets himself fall.

(b) In labyrinthine lesions the direction of falling with the patient in the Romberg position bears a direct relation to the plane of the nystagmus. Bárány has elaborated certain rules to show how the attitude of the head and the quick phase affect the fall; but as we pointed out some years ago there is a general rule for all positions of the head, namely, that the patient falls in the direction of slow phase of the nystagmus. And as we have shown even with the cerebellum injured the labyrinthine reaction, nystagmus, attitude and falling, can be elicited by labyrinthine stimulation. As Bárány pointed out there is not the same connection between the direction of nystagmus and the direction of falling in cerebellar affections.

(3) Lewandowsky says that the particular character of cerebellar ataxia is the disturbance in standing and walking, "Störung der Aufrechten Haltung und des aufrechten Ganges" (loc. cit. p. 842). This results in those oscillations, that drunken gait, the titubation vertigineuse of Duchenne, so characteristic of cerebellar disease and not seen in labyrinthine lesions. Further, the cerebellar patient shows dysmetria; his movements lack their proper proportion or measure in time and space; they are too rapid and too abrupt and miss their aim; the initial impulse is too strong, the speed too great, the arrest too late.

The ataxia apparent after labyrinthine removal is due to a failure of the proper adjustment of the muscles necessary to the maintenance of equilibrium and orientation, owing to a failure in the accurate perception of the change of aspect of the head in space. To put it briefly we might say that where the cerebellar ataxia is dynamic the labyrinthine is chiefly static.

(4) In labyrinthine disease the physiological action of the labyrinth can be shown to be lost by the usual caloric and rotation tests. This fact, I feel is so appreciated by you that I do not intend to dwell on it.

We have, therefore, in pure labyrinthine and pure cerebellar cases sufficient data at our disposal to decide the question. It is difficult in the mixed cases. If, however, we can exclude with these data the purely local labyrinthine and the purely local cerebellar we have gone some distance to locate the lesion, and can proceed intelligently to cases that involve both structures.

Discussion.

Dr. E. B. Dench, New York City, cited a case illustrative of the difficulties of diagnosis in lesions of the cerebellum. The patient was a very sick man, with a history of three weeks' standing when he began to have headaches, and in two weeks paralysis of the right external rectus. The blood showed a polymorphoneuclear leucocyte count of 80 per cent. He was called in to confirm or exclude the diagnosis of cerebral abscess. The man was blind in the right eye and the right static labyrinth was dead. Hearing impaired but not lost on right side. The left labyrinth was active to the caloric test but sluggish. With the paralysis of the right external rectus and a dead static labyrinth on the right side and the acoustic labyrinth active, he disregarded the old cicatricial drum membrane upon the right side and made the diagnosis of tumor of the cerebellum. The man died. At autopsy a large cyst was found in the left cerebellar fossa, but encroaching upon the right cerebellar fossa.

Dr. George E. Shambaugh, Chicago, noted that the essayist had apparently taken no note of the phenomena of labyrinth tonus and had not even used the word tonus. The more the speaker studies the phenomena arising from disturbance of the vestibular labyrinth the more he is inclined to believe that the chief function of the vestibule is to provide tonus to the skeletal muscles and in this way to assist in the function of equilibrium. The function of supplying data which gives us our sense of turning is relatively unimportant. If he understood Dr. Wilson correctly, the nystagmus arising from disease of the labyrinth is always directed towards the unaffected side. As a matter of fact this is true for about all of the cases which come under observation, but the reason probably is that the depression of labyrinth tonus in the affected ear follows so quickly on the development of labyrinthitis, thus permitting the normal tonus from the opposite ear to produce the unbalance which results in nystagmus directed to the normal side. On the other hand, cases of acute labyrinthitis are occasionally observed where in the onset a nystagmus is produced which is directed towards the affected side. The speaker has explained this phenomenon as the result of an increase in the normal tonus from a labyrinth in the early stages of inflammation, where a congestion alone and before an inflammatory exudate has filled the labyrinth, producing a suppression of function. As soon as a cerebellar abscess develops it is interesting to note that, as a rule, a nystagmus is produced which is again directed towards the affected side, just as though the cerebellar abscess were producing a unilateral increase in tonus by stimulation of the trunk of the vestibular nerve.

Dr. B. Alexander Randall, Philadelphia, thought the differentiation between the irritative and the depressive influence upon the labyrinth a mere hypothesis. In the hyperemic stage of labyrinthitis there was an over-production of endolymph, for we have in the attached area of the semicircular canal, as in the striavascularis of the cochlea, an organ for its secretion. Hence there was a flow of the en-

dolymph towards the vestibule with the same mechanical stimulation as in the turning or caloric tests.

Dr. Wilson, in closing the discussion, said he had nothing further to add to the explanation of the quick phase of nystagmus directed towards the side of the lesion. He wished to emphasize the point that experimentally the direction of vestibular nystagmus could be altered by pressure on the nerve. It had been asked why no notice had been taken of tonus. Dr. Wilson had omitted references to it for several reasons. Thus there is no means of measuring tonus, which normally is very fleeting in its manifestations. There are many centres outside the labyrinth which influence muscle tonus, thus alterations in tonus are very frequent in cerebral and cerebellar disease. To depend on an appreciation of tonus in a differential diagnosis of labyrinthine and cerebellar disease would be hopeless. It appeared to him that too much had been made of it as a special labyrinthine function.

A BLOODLESS METHOD OF ENUCLEATING TONSILS.

By E. TERRY SMITH, M. D., Hartford, Conn.

To do a careful surgical operation one of two conditions must obtain,—either the operative field must be continually plainly visible, or the operation must be done by the sense of touch. Operating in the dark is dangerous and unsurgical. In describing the method of enucleating tonsils which we have employed for the last three years, I make no claim to originality, but we have had such uniformly good results and such slight loss of blood, that it may be of interest to others.

After deciding that the tonsils are sufficiently diseased to be removed, the operation should be considered as a major surgical procedure. The patient should be operated upon at a hospital, and gas-oxygen-ether should be the anesthetic of choice, and should be given through the Davis-Crowe mouth gag. Personally I prefer the recumbent position with the head slightly lower than the rest of the body. An electric headlight is the best form of illumination, and one should have a competent trained assistant. Since we have been doing our present operation we have not had a disturbing tonsillar hemorrhage. We use a suction apparatus to remove the blood and mucous from the throat and, in the great majority of cases, control any bleeding by pressure. We are always prepared to tie any bleeding points and, if necessary, to sew the anterior and posterior pillars together. We also have a Michel's clamp which has never been used. These little silver hemostats, originally made for Dr. Cushing, and modified for tonsil work by Dr. Crowe, we have found very useful in a number of cases. They are much quicker than tying, and we have known of no bad results from their use. Hemorrhage is caused chiefly by injury to the walls of the sinus tonsillaris, which is inexcusable except in cases where the adhesions are very dense. Infection is also frequently caused by leaving a ragged, sloughing sinus tonsillaris. When one examines

the tonsil after its removal, it should not be covered with parts of the superior constrictor of the pharynx.

One should use great care not to injure the posterior pillar or the palatopharyngeus muscle, as any injury that leaves the palate less free than normal in its backward and forward play has a deleterious effect on the voice.

Perhaps it may be well to give in detail our routine in operative work on the tonsil. All cases are sent to the hospital; history is taken, especially with regard to hemophilia; physical examination is made, (including examination of urine and blood coagulation test). If the blood coagulates within ten minutes, I think it is perfectly safe to operate.

We have the usual preparation for a major surgical procedure, and all adults are given a hypodermic of morphine 1-6 gr. and atropine 1-150 gr., twenty minutes before the operation. We use as an anesthetic gas-oxygen-ether.

The photograph on page 500 shows the usual set-up for the operation. The anesthetist is at the patient's left side holding the Davis-Crowe mouth gag. Only slight pressure should be applied to the gag, to avoid wounding the tongue. The assistant stands back of the patient's head with both hands free so as to be able either to hold instruments, use suction apparatus, sponge, or control hemorrhage. The operator stands at the patient's right and wears an electric headlight. The instrument stand is at the operator's left. A clean nurse should be at the assistant's right to pass sponges, etc. The patient being in a recumbent position, with the head slightly lower than the body, the mouth gag is introduced carefully so as not to injure the teeth and is opened wide enough to give a good view of the operative field. (Dr. O. R. Witter's suggestion to tell the patient while going under the anesthetic to keep the mouth open, we have found useful). The patient should not be very deeply anesthetized. This is facilitated if gas is given preliminary to ether.

First Steps of Operation. Tonsil forceps are firmly pressed against the tonsil while closed, are then opened, and a generous amount of tonsil is grasped. (The forceps I use were originally made by Vaast, of Paris, and were recommended

to me sixteen years ago by Professor Luc, of Paris. They are now made by J. E. Kennedy & Co., of New York). The tonsil is now gently drawn from its bed out into the throat, and an incision is made through the plica, extending from the middle of the anterior surface of the tonsil to the base of the tongue, care being used not to injure the capsule. Another incision is then made from the beginning of the first incision up over the tonsil and extending down on its posterior surface, care being taken not to injure the palatopharyngeus muscle or the posterior pillar.

Second Step. The tonsil is separated from the anterior, and the upper part of the posterior pillar by means of the Carter knife.

Third Step. (Left Tonsil) The anterior pillar is retracted by a Hurd's retractor and held by an assistant, while another pair of forceps is applied to the upper part of the tonsil to help evert it from its bed. Traction is applied to the tonsil and it is dissected by blunt dissection from the sinus tonsillaris, care being taken not to injure the capsule.

Third Step. (Right Tonsil) The procedure is the same as with the left tonsil except that the anterior pillar is retracted by operator, and traction on tonsil is applied by assistant, while operator dissects tonsil by blunt dissection.

Fourth Step. The tonsil now being almost entirely freed, the snare (Beck-Schenck tonsillectotome modified by Dr. C. H. Borden) is applied to the base, and the tonsil removed.

Fifth Step. Sponge wet with adrenalin 1/1000 is introduced into sinus tonsillaris and firm pressure made for three or four minutes.

Sixth Step. Retraction of pillars and inspection of sinus tonsillaris for evidence of hemorrhage or remains of tonsil.

If the operation is done carefully, very little bleeding will be experienced, the pillars of the tonsil will not be injured, and after the throat becomes healed it will be perfectly normal in appearance and not in the slightest degree distorted. We have operated on the throats

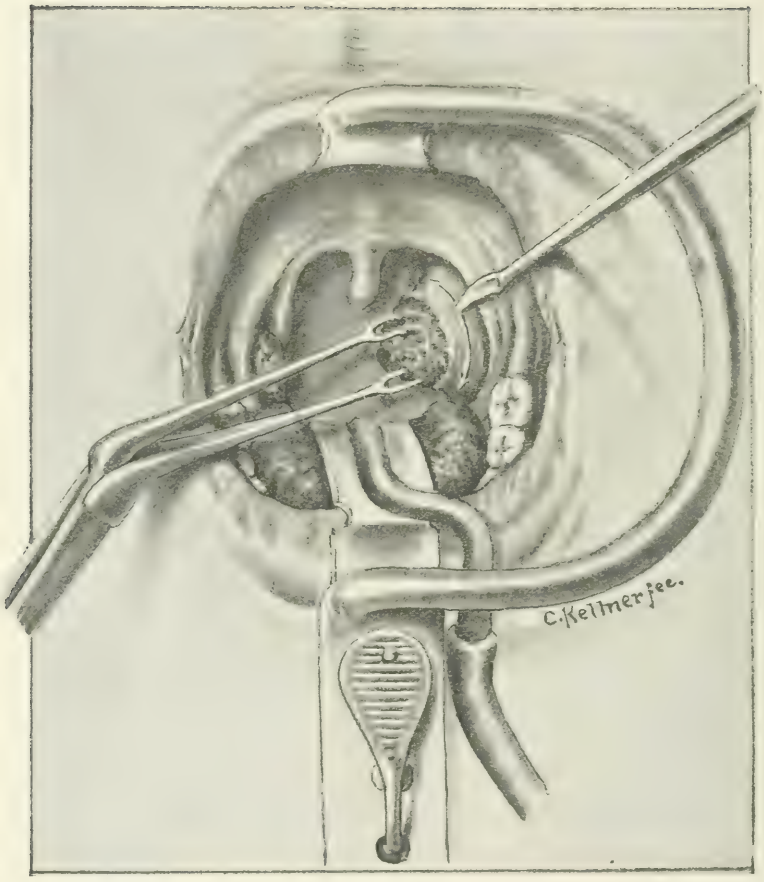
of a number of singers and in every case the voice has been improved by the operation.

The instruments we use are as follows:

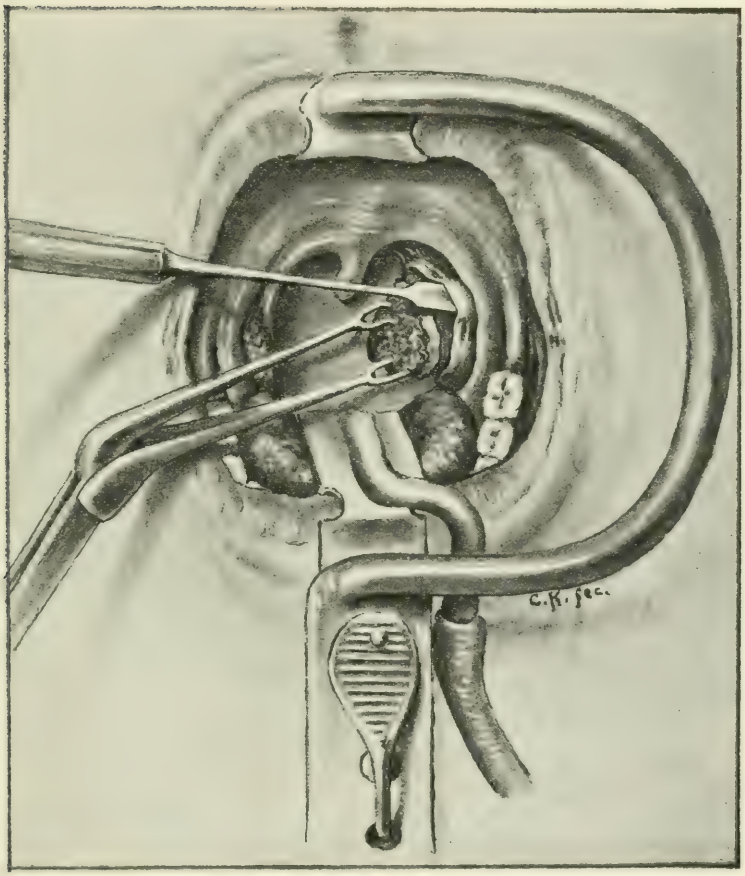
1. Metal tip for suction apparatus. (Designed by Dr. C. H. Borden).
2. Prince's tonsil scissors.
3. Needle for suturing pillars together.
4. Shallcross's double curved hemostatic forceps.
5. Michel's metal clamps.
6. Forceps for placing Michel's clamps.
7. Forceps for removing Michel's clamps.
8. Forceps for placing the Cushing-Crowe clamps.
9. Small silver clamps for bleeding points, Cushing-Crowe.
10. Schoemaher's artery forceps.
11. Beck's tonsil hemostat.
12. Tonsil forceps, originally made by Vaast, Paris.
12. Tonsil forceps, originally made by Vaast, Paris.
13. Hurd pillar retractor.
13. Hurd pillar retractor.
14. Carter knife.
15. Abraham's tonsil knife.
16. Headlight.
17-b Small tongue depressor for ether gag.
17-a Medium tongue depressor for ether gag.
17. Davis-Crowe mouth gag, large tongue depressor. (Made by Charles Willms Surgical Instrument Company, Baltimore).
18. Sponge forceps.
18. Sponge forceps.
18. Sponge forceps.
18. Sponge forceps.
19. Tonsil snare. (Beck-Schenck tonsillectotome, modified by Dr. C. H. Borden).
19. Tonsil snare. (Beck-Schenck tonsillectotome, modified by Dr. C. H. Borden).

After Care.

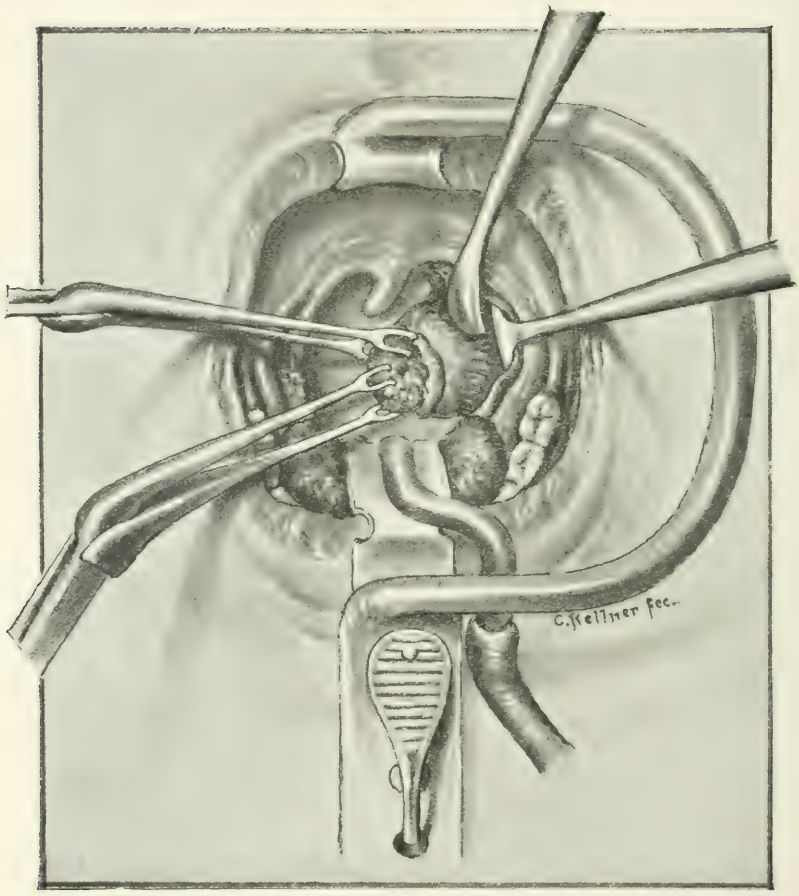
The throat should be kept clean with an alkaline mouth wash; sinus tonsillaris should be touched up with five per cent solution of silver nitrate p. r. n., until throat is healed, which usually takes from one to three weeks. Patient's general condition indicates length of stay necessary in hospital; the usual time is from one day to a week.



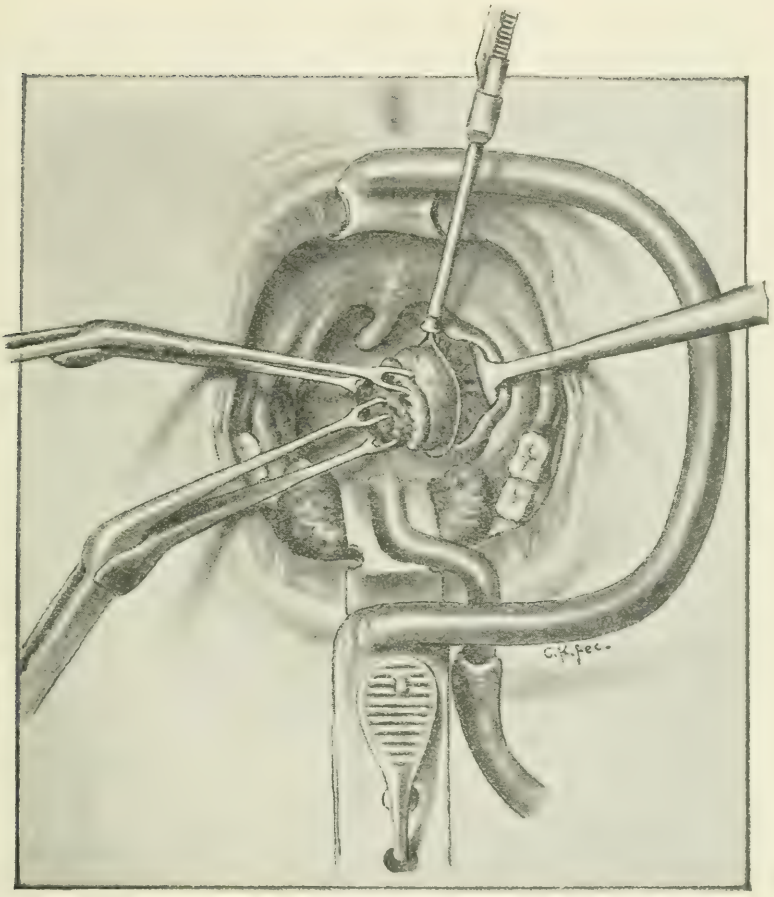
1st Step.



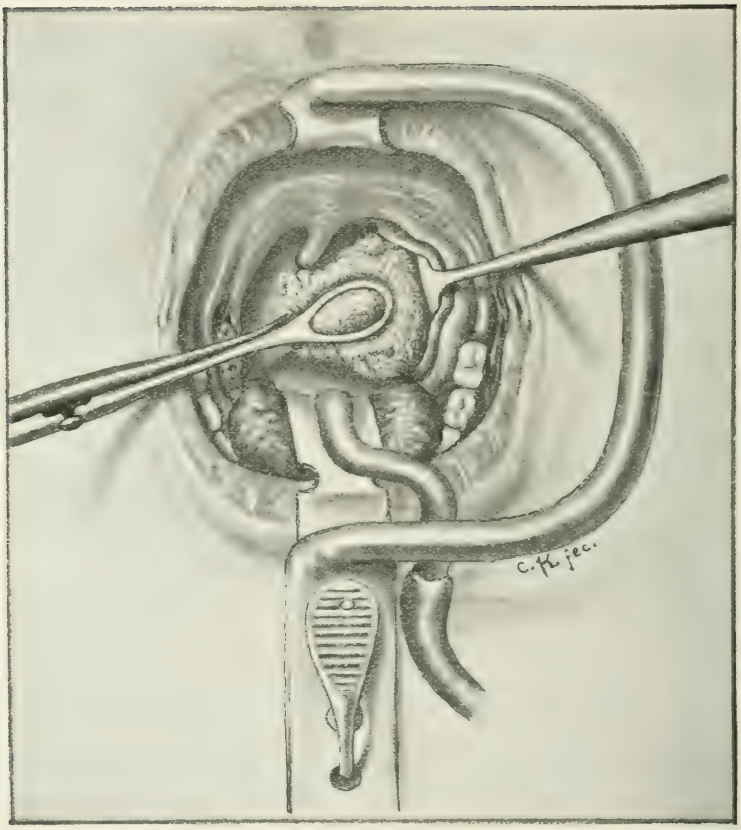
2nd Step.



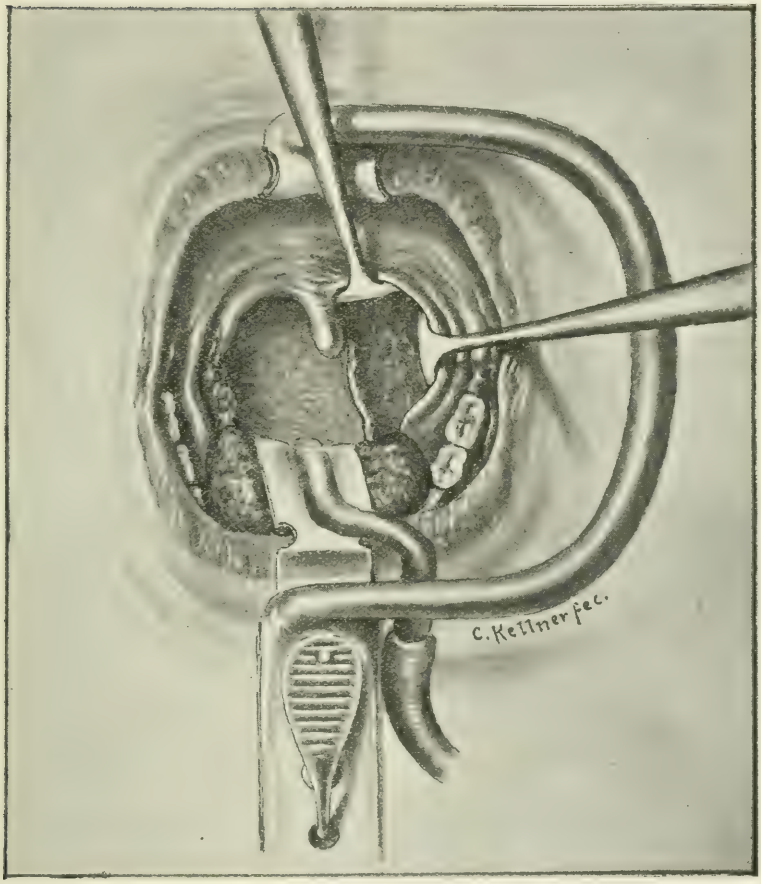
3rd Step.



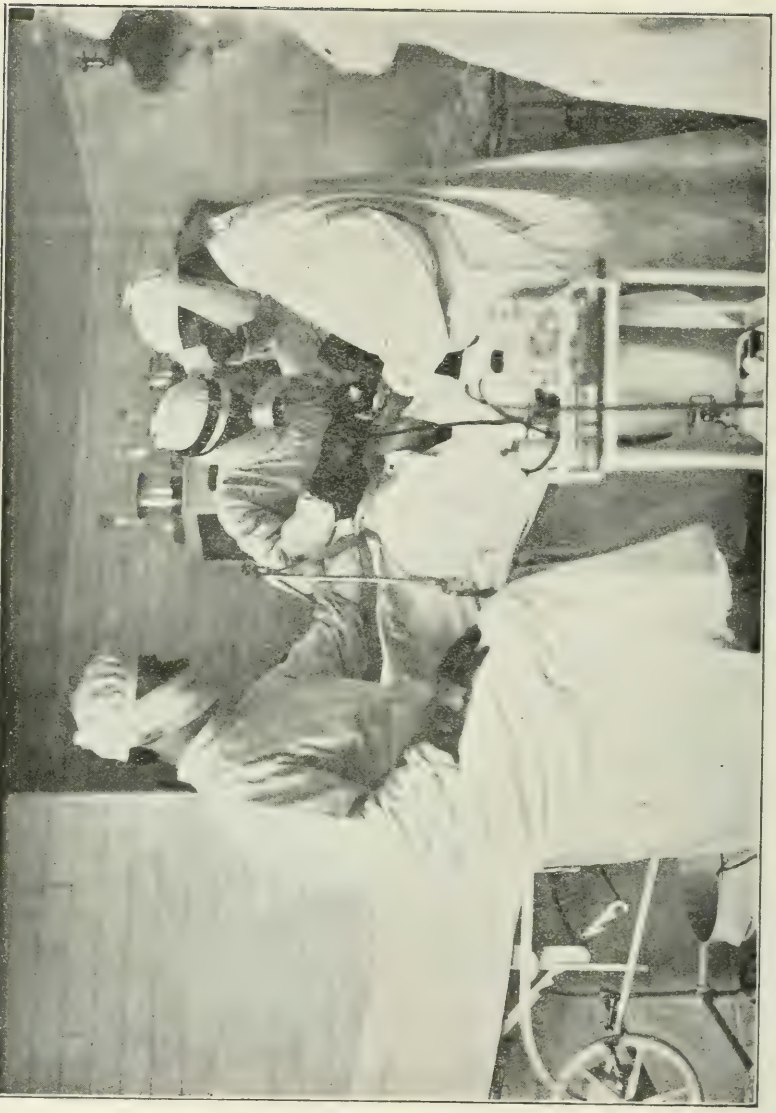
8th Step.



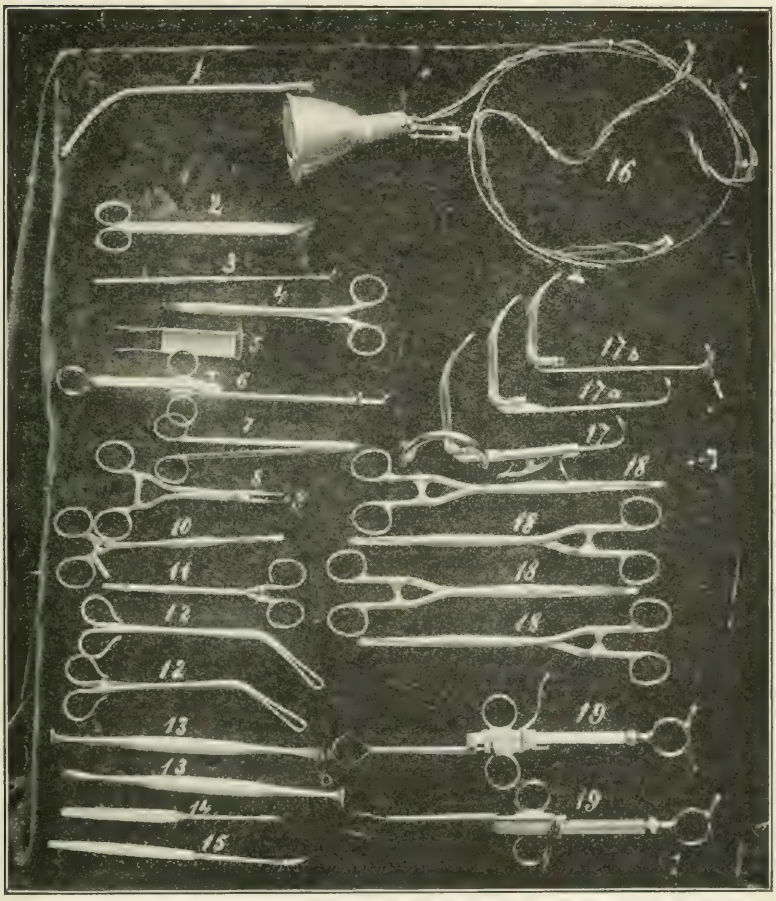
5th Step.



6th Step.



Set Up for Operation.



Instruments.

Discussion.

Dr. George E. Shambaugh, Chicago, objected to the use of metal clamps in the fauces because of the danger of inhaling in the bronchi a loosened clamp.

Dr. Emil Amberg, Detroit, Mich., asked the essayist why he used adrenalin. He called attention to the effects of adrenalin, not only with reference to post-operative hemorrhage, but also with regard to its absorption. It had been shown experimentally that a comparatively small amount of adrenalin would kill an animal. In view of this he would be afraid to use it on any surface where it might be absorbed in large quantities.

Dr. Smith, in closing the discussion, said he had used metal clamps up to two and a half years ago, since which time he had used nothing but pressure. He had used adrenalin invariably except in a series of fifty cases, in which he had tried other solutions. He had never noticed any ill effects from its absorption. He always stopped the hemorrhage before it came, so to speak.

DECOMPRESSION FOR THE RELIEF OF CHOKED DISC FOLLOWING THROMBOSIS OF THE LATERAL SINUS.

By HARRY FRIEDENWALD, M. D., Baltimore, Md., and J. W. DOWNEY, Jr., M. D., Baltimore, Md.

Choked disc is not an unusual occurrence in thrombosis of the lateral sinus and is mentioned in all works upon the subject. Kipp observed it in 8% of his cases (Z. F. O. Vol. VIII) and Jansen in 50% (A. F. O. Vol. XXXVI); numerous writers have reported cases in which edema of the optic nerve was observed during the course of a lateral-sinus thrombosis or following the ligation of the internal jugular vein for the cure of that condition. Until recently a satisfactory explanation has not been given for this otitic complication; nor have we had any guide for the relief of the choked disc and the prevention of the optic atrophy and the resulting loss of vision which is certain to follow.

A case occurring in the practice of one of the writers and reported some time since, (Journ A. M. A. Vol. 51, p. 483) is illustrative of the ordinary course and termination of these cases. A brief history of this patient is as follows:

The patient, a colored woman, aged 32, was first seen on February 20, 1906, for acute suppurative otitis media and mastoiditis on the left side. The patient did not consent to an operation until March 3. There was pus under some pressure in the mastoid cells and the bone was soft. The lateral sinus was exposed for about one-half an inch; part of it was found covered by granulation tissue. The tip of the mastoid was one large cell filled with pus and granulation tissue on the posterior part of which the sinus was likewise exposed. After packing the latter cavity and withdrawing the packing, there was a profuse hemorrhage from the lateral sinus, though no violence had been used. It was controlled by packing with iodoform gauze. Her temperature before the time of the operation was about 99 F. Pulse 80 to 98. On the following

day the temperature was irregular, reaching 103.6 F. on the fourth day after the operation, while at the same time her pulse dropped on the second day after the operation to 64, and remained below 70 for several days, except when the temperature was very high and then the pulse rose to 100.

On the fifth day (temperature between 98.8 F. and 100.5 F.) abducens paralysis set in and mild optic neuritis was seen. From this period on the temperature remained normal, though the pulse continued slow during the entire first week, but the neuritis increased and rapidly became a typical choked disc with hemorrhages in both eyes, more marked in the left.

Seven months after the operation, there was very little swelling of the discs, the vein slightly tortuous, but not very full, the arteries somewhat narrow, the color of the discs pink, and margins obscured; the condition of the left eye being more marked than that of the right eye. V. R. E., 16/30; V. L. E., 10/100.

In this case, which was attributed to serous meningitis, we interpret the choked disc as resulting from the thrombosis of the lateral sinus, following injury of its ulcerated wall and the firm packing required to control the hemorrhage.

In view of the serious damage to vision in these cases, we determined to avert it, if possible, in the following case which, in the symptoms and early course, greatly resembles the first. When choked disc became definitely established, subtemporal decompression was performed. The history of this case and the result of the operation are as follows:

Eliza C., colored, 25 years old, was admitted to the Baltimore Eye, Ear and Throat Hospital, February 13, 1914, having been ill for ten days with discharge from and severe pain in and around the right ear. Her temperature was 103° on admission. She was extremely tender over the mastoid antrum and tip, and there was a profuse sero-purulent discharge from the right ear. The patient was operated on immediately for acute mastoiditis, and the operation disclosed nothing out of the ordinary. The mastoid was of the large cellular type and was filled with pus and granulations. The dura was not exposed at any point. The patient was returned to bed in good condition

and her temperature on the following morning was 99°. She was not entirely comfortable, however, and by three o'clock in the afternoon of February 14th, her temperature had again risen to 104°. The wound was reopened and irrigated and the temperature came down to 100°, but by the following afternoon there was another sharp rise to 104°. There were chilly sensations and the patient was undoubtedly septic. The temperature and the symptoms became more and more clearly those of lateral sinus thrombosis. On February 20th, (six days after the mastoid operation) the internal jugular vein of the right side was ligated and the lateral sinus exposed and opened. This operation, likewise, presented no unusual features. The internal jugular was smaller than usual and the glands along its course were enlarged. The sinus, when exposed, was slightly flattened but not collapsed, and bled freely when opened. The condition was that of beginning thrombosis, or rather phlebitis of the lateral sinus.

The patient made an uneventful recovery, the temperature returning to normal by the third day, following which there never was a rise of temperature above 100°. *This almost normal course of the temperature is of extreme interest in view of the later complications in the case.* Cultures made at the time of the mastoid operation were negative. Later cultures, from the mastoid wound, gave a faint growth of the *streptococcus mucosus.*

Although the patient seemed to be progressing well, she was never quite comfortable and complained a great deal of sore throat and headache.

Four days after the operation she had severe headache in both the frontal and occipital regions. Six days following the operation, she complained of poor vision. An ophthalmoscopic examination made at this time disclosed blurring of the edges of the optic nerves, and fullness of the veins of the fundus oculi. The condition was more marked in the right eye than in the left. For two days, slight edema had been observed in the neck and the right side of the face, most marked around the eye, this was transient and had disappeared on the sixth day after the operation. The swelling

of the optic nerve increased rapidly, the papillae becoming markedly elevated, the retina edematous around the nerve entrances and the veins enormously dilated and tortuous. On March 4th, small hemorrhages were noted in the right retina. The choked disc was at all times more marked in the right eye than in the left. Vision tested at this time showed R. E. 16/40; L. E. 16/60., and it was found the same at all examinations made before the decompression operation. The visual fields for white, blue and red were concentrically contracted about ten degrees in each eye. From March 7th on to the time of the decompression there were no changes noted in the condition of the eyegrounds. A complete neurological examination, made at this time, disclosed nothing of interest. The Wasserman reaction was negative. The blood pressure was equal to 134 mm Hg. The leucocyte count, made several times, was normal (from five to six thousand) and the differential count showed normal proportions. Urinalysis showed nothing abnormal.

The patient complained continually of *headache*, more severe when she sat up, located both in the frontal and occipital regions. Her temperature chart showed but slight variation from the normal; there was usually a rise to 99 in the afternoon; the temperature rarely reached 100 and never rose above. The average pulse rate was 80, and it was never below 70, rarely above 90.

On March 14th, (three weeks after the operation upon the sinus) a subtemporal decompression was performed by Dr. Charles Bagley, Jr., who has given us the following notes of his findings:

"March 14th, 1914. Right subtemporal decompression. The usual approach was made through the temporal muscle, and a fairly liberal bony opening made. The dura, when exposed, was found to be tense, and when incised permitted cerebrospinal fluid to spurt freely. There was a great excess of fluid and bulging of the cortex. After evacuation of a considerable amount of the fluid, the cortex ceased bulging. The wound was not drained. The closure was made in the usual manner. Operative recovery was good."

From the time of the decompression operation, the patient made a rapid recovery. The choked discs rapidly subsided, the hemorrhages in the right fundus were absorbed and the veins resumed their normal calibre. The patient no longer complained of headache, and on her discharge from the hospital, April 1st, her vision had improved to R. E. 16/20; L. E. 16/30. Three months later, it was impossible to find any ophthalmoscopic evidence of previous disease of the optic discs and her vision was 16/16 with each eye.

At the time of this operation we were ignorant of any similar reports. We have since, however, found the article of Auerbach and Alexander (Mittheilungen A. D. Grenzgebiet der Med. U. Chir., 1913, Vol. 25, p. 431) in which they describe a similar case, in which the decompression operation was performed. The article is likewise important for its references to other unoperated cases, and for the thorough discussion of the probable factor in lateral-sinus thrombosis responsible for the choked disc.

Auerbach and Alexander's case was that of a man 26 years of age who had had cholesteatoma of the left ear for years. He was operated on for lateral sinus thrombosis with choked disc, the symptoms having appeared five days previously. There were cholesteatomatous masses, perisinus abscess and a fistula leading into the lateral sinus. The internal jugular was tied and the diseased sinus completely opened. The temperature fell within the next few days, but the choked disc increased and vision decreased so that the patient became completely blind in the right eye and almost blind in the left. The retina showed the appearance of an albuminuric retinitis but the urine was normal. Three days after the operation, a neurological examination disclosed a paresis of the right abducens and the right facial nerves. There was no headaches, no vomiting, no slowness of the pulse. Diagnosis: Meningitis serosa, and, therefore, it was determined to puncture the right lateral ventricle from Keen's point. The result was negative. The wound was enlarged backwards and the cerebellum was ex-

amined for abscess but without result; there was no improvement in the condition.

Eleven days later, a left subtemporal decompression was performed and on the following day, the pupils responded to light. Eight days later the patient could count fingers at 1 m. and in another week, the vision was R. E. 1/15; L. E. 1/4, with a decrease in the swelling of the disc and the absorption of the extravasations of blood.

The patient died three and a half months later from an erysipelatous infection of the decompression wound.

The authors recognize the great difficulty in explaining choked disc in lateral sinus thrombosis, especially as the choking does not decrease after a successful operation but increases. They refer to Bartels, who has reported such cases and was unable to give a satisfactory explanation. (Zeit. f. Augenh. Vol. XXI, p. 23.)

Bartels' cases resemble the case of Auerbach and Alexander. The choking of the disc was not as marked before operation and reached the climax in the second to the fifth week after the operation, and not in a few days after the ligation of the jugular. The vision was not so markedly reduced, and only in one case was there a paresis of the facial and abducens nerves.

Auerbach and Alexander incline to the mechanical explanation of choked disc in cases of sinus thrombosis as the result of interference with the venous outflow. They refer to the work of Rüdinger, who has shown that the superior longitudinal sinus in more than half the cases turns to the usually larger right lateral sinus while the straight sinus commonly turns to the left. The two lateral sinuses are unequal as are the internal jugulars. These facts are corroborated by Macewen.

Spalteholz described the superior longitudinal sinus as frequently emptying its larger right branch into the right lateral sinus, its smaller left branch into the left. Almost as often it empties itself undivided into the right lateral sinus—very rarely into the left. The straight sinus at times enters undivided into the left lateral sinus, but it generally

divides, its larger left branch emptying into the left, its smaller right into the right lateral sinus. Toldt and Henrica and Kikuchi have likewise studied these variations with similar results.

In their own case, Auerbach and Alexander, assume that the closure of the left lateral sinus resulted in interference with the venous outflow from the interior of the brain, with a damming up of blood and with consequent edema of the brain, increase of intracranial pressure and its resulting symptoms.

Ruttin gives a similar explanation (Laryngoscope, Vol. XXI, p. 1051, Verhandl. Otol. Gesellsch., 1911, and Arch. f. Ohrenheilk, 1912, Vol. 88, p. 22). He thinks that when the right sinus is closed that the surface circulation suffers to a lesser degree than when the left is closed. The choked disc is less marked and altogether milder.* That tying of the right jugular vein in cases where it is much larger than the left may produce fatal cerebral edema has been shown by Linser (Beitr. z. Klin. Chir., Vol. 28, p. 642). He found two such cases reported and added one of his own and he likewise referred to the experiments of Reisinger (Zeits. f. Heilkunde, 1882).

It appears to the writers, that the explanation of venous engorgement is most satisfactory and that the great variation in vascular distribution accounts for the occurrence of choked disc in one case and its absence in others.

In our first case we are not of the opinion that there was a thrombosis of the lateral sinus before the operation, and

*Ruttin further thinks that choking of the disc occurs with much more frequency in thrombosis of the lateral sinus than in any other pyogenic brain condition. In twenty-three cases of temporal abscess, he saw choked disc in only three of the cases. In twelve cases of cerebellar abscess, he found choking in two cases, but in thirty cases of sinus thrombosis, he found eight with choked disc. He attributes the choking in cases of cerebellar tumors to the same mechanical obstruction as that causing the condition in lateral sinus thrombosis, viz an engorgement of the cavernous sinus as the result of a regurgitation of blood from the superior and inferior petrosal sinuses, in turn cut off by obstruction in the laternal sinus which may be due to pressure, as in the case of tumors, or a thrombus. He is sure that serous meningitis did not exist in his cases, and therefore, thinks that the condition of the optic nerve, and the facial and abducen paresis sometimes seen, is undoubtedly due to mechanical causes, and this is further substantiated by the occurrence of edema, unilateral headache and other symptoms of engorgement. Why it should occur in one case and not in another can only be conjectural but may be best explained by the anatomical difference which undoubtedly exist in different individuals.

in the second, symptoms of thrombosis did not arise until several days after the mastoid operation. Unfortunately, no ophthalmoscopic examination was made before the signs pointing to visual disturbance appeared. But when first seen, the ophthalmoscopic changes were evidently only beginning and their rapid increase was subsequently watched day by day, therefore, the thrombosis in the lateral sinus may but partially interfere with the current of blood—and the complete stoppage may occur only when the jugular vein is tied; this was probably the case in our second patient.

We do not believe that there was a meningitis in either of the cases we have reported. The temperature, the pulse and the general clinical conditions would seem to contradict the existence of any inflammation, however mild. The condition of cerebral and meningeal edema should, therefore, it appears to us, not be termed serous meningitis. In some cases there are other signs of venous stasis besides those in the eyegrounds; in our second case, there was definite edema of the face and neck. Also, the very satisfactory result of the decompression operation in our second case and in Auerbach and Alexander's is still more convincing proof of the non-inflammatory character of the edema. Aside from the explanation they give of an interesting condition, these cases serve as a valuable guide in dealing with it in the future.

THE EAR-TESTS OF BARANY IN LOCATING CEREBELLAR AND OTHER ENCEPHALIC LESIONS.

By B. ALEX. RANDALL, M. D., and ISAAC H. JONES, M. D.,
Philadelphia, Pa.

The importance to the aural surgeon of the Barany tests is generally known, and they have even been overworked in leading to needless operations upon the labyrinth; but in the broader aspects of diagnosis we have probably made only a beginning, and it is to this side of the matter that we would claim the attention of the American Otological Society. For it is the otologist who alone can rightly make some of these investigations, since the neurologist and the general surgeon rarely have the needed otological training: and the otologist, for that matter must renew or enlarge his Neurology if he is to progress accurately in this complex field. Yet time for successful intervention in many intracranial morbid conditions is usually short and may not permit of any elaborate cooperative study by a group of specialists—success will be highest where competent head and hand can be united in one person, as we have seen in the low mortality of Lateral Sinus Thrombosis when operated on by the skilled aural surgeon.

With so good a leader as Barany in this modern advance, progress has been rapid and generally sure; yet errors have crept in at many points and obtained temporary currency; and only the combined critical study, tested and corrected by the crucial touchstone of operation or autopsy, can convert the findings from empiric gropings into scientific data leading on to new conquests. For this the large hospitals offer fine opportunities, and we have been very fortunate in Philadelphia to have been able to utilize these to a notable extent.

With this purpose there has been created in the University of Pennsylvania a Department of Neuro-Otology, devoted ex-

clusively to this work; and in the past year and a half we have examined 125 pathological cases.* While giving full heed to the aural aspects, with treatment of any local conditions requiring it, study has been especially directed to the intracranial side of the subject, guided and aided by Drs. Mills, Weisenburg and others on the neurological side and by Dr. C. H. Frazier on the surgical. For this purpose the pointing reactions have far surpassed the nystagmic; and most of our stress will be placed in this paper on these later developments of the Barany testing. Of the intra-cranial cases eighteen have come to operation and four to autopsy. These sections, ante-mortem or post-mortem, have shown the deductions from the ear-examinations to have been remarkably accurate, and have led us to believe that this work is valuable and worthy of this preliminary report.

As usual in such complex and many-sided researches, anomalous or contradictory findings frequently confront us. Physiologic phenomena, not to speak of the psychic, do not group themselves so readily under simple laws as do the merely physical. Yet some inconstancy of findings, even in the same case, marks the vital characteristics of our subject —we are dealing with advancing processes not with fixed morbid lesions; and the variations may be most instructive when understood, however disconcerting they may at first appear to one who vainly undertakes to demonstrate a reaction previously present.

It is hardly necessary to tell this Society the technic of examination. In brief, however, as regards the pointing reactions, the normal person is always aware of the location of his hand or finger in space when his eyes are closed; and he is able with it to find an object previously located by him, as for example, the finger of the examiner held in front of him. With the ear stimulated, either by cold or hot water or air or by turning in a smoothly revolving chair, he is no longer able to find the finger, but points *past* it in a definite

*Also many more not found pathologic; although we cannot emulate a colleague who claims—"I apply the labyrinth-tests to every case coming to the clinic."

direction, either to one side or the other, above or below, depending upon the exact stimulation employed. These "past-pointing" reactions are absolute in the normal; their modification marks the abnormal, and challenges us to find the explanation and locate the fault.

If the right ear is douched with cold water of a temperature of 68° F., the normal person points to the right; if the right ear is douched with hot water of 112° F. he points to the left. This is true, in both instances, also of the wrist, elbow, foot, head and trunk. Or if the patient is turned in a rotating-chair towards the right and then suddenly stopped, all the pointings will be towards the right; and *vice versa* after turning to the left. In each of these tests the head must be rigidly supported in the chosen position.

There has been so much needless confusion, however, in the descriptions of these tests, especially in stating the direction of the nystagmus in comparison with the past-pointing, that perhaps we may venture to suggest an absolutely reliable and easy method of remembering these facts, namely:—the usual routine methods are by cold water and turning: now if to the right, (that is either turning to the right or douching the right ear) everything is to the right—all the pointings, shoulder from above, shoulder from below, elbow, wrist, neck or trunk, and the falling reactions, all are to the right. Now the deviation of the eyes, just as all the other reactions, is also to the right: but this is the slow movement of the eyes to the right. The "recovery," the quick movement to the left, the cerebral component, is more conspicuous, however, and therefore this phenomenon has been called "nystagmus to the left." *Vice versa,* to emphasize this:—if the left ear is douched or the patient is turned to the left, all reactions are to the left, and the true vestibular movement of the eyes is also to the left, although mistakenly the nystagmus is *spoken of* as toward the right. This makes it very simple, especially in teaching students; but to tell them, as does Barany, that the past-pointing is always in a direction opposite to the nystagmus, is not only confusing but

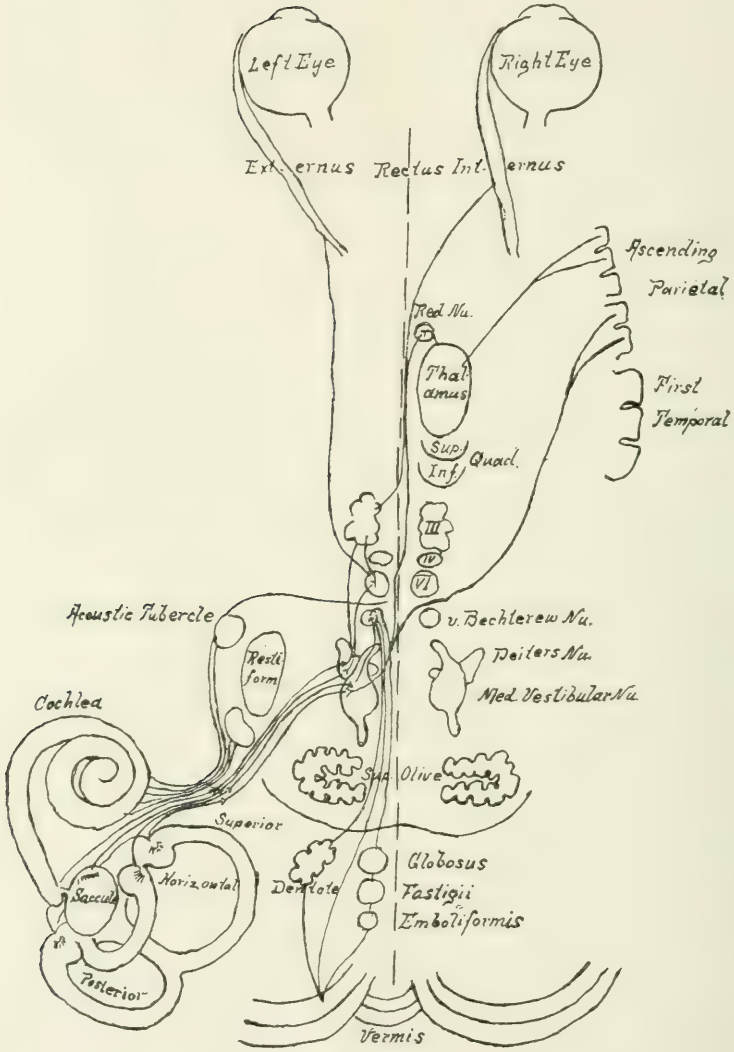

Left Eye
Right Eye
Ext. ernus
Rectus Int. ernus
Ascending
Parietal
Red Nu.
Thal amus
First
Temporal
Sup.
Inf.
Quad.
III
IV
VI
Acoustic Tubercle
v. Bechterew Nu.
Resti form
Deiters Nu.
Med. Vestibular Nu.
Cochlea
Sup. Olive
Superior
Horizontal
Saccule
Globosus
Dentate
Fastigii
Emboliformis
Posterior
Vermis

Figure I.

misleading: simply tell them:—if to the right, everything is to the right; if to the left, everything is to the left.*

As to the reasons for these reactions:—first, as regards the nystagmus, this seems easy and fairly understood. The stimulus travels from the ear to the muscles of the eyes producing a movement to one side; the quick re-adjustment in the opposite direction is the cerebral attempt to overcome the abnormal movement.

Second, a normal person past-points either to the right or the left, according to the stimulation, because he has a disturbed sensation of his position in space.

Now for a normal person to point, under stimulation, in any direction, there must be a definite arc; just as there must also be a definite tract from the ear to the eye, in order to produce nystagmus. In addition, if douching the ear or rotation produces vertigo, it also naturally follows that there must be an arc from the ear to the subjective centers, which can perhaps be best spoken of as the subjective arc. If therefore, there should be after stimulation an absence of nystagmus, an absence of past-pointing or an absence of vertigo, we can assume an interruption or lesion along the line of the arc concerned. This is at the basis of this matter of spleen-cephalic localization. The ear is stimulated, the impulse is carried by means of the VIII Nerve to a group of nuclei in the lateral part of the medulla, namely the Deiters' nucleus, triangular nucleus and Von Bechterew's nucleus; from here the impulses go in a number of directions. One set goes from the nuclei to the posterior longitudinal bundle and from here to the VI, IV and III nuclei in the pons and crus, thus causing a nystagmus: this is definite and in all probability accurate.

As for the cerebellar circuit, it may take many years to work it out positively. Our present conception is that on each side the fibres from the *horizontal* canals go to the

*Is it not time to select a substitute for this misleading word "nystagmus," now hopeless, unless in a general sense, in this connection? In anesthesia or stupor the reaction is solely a conjugate deviation. Why not call it the nystagmic deviation?

Deiters' group, thence to the cerebellum by way of the juxta-restiform body (which is the internal portion of the inferior cerebellar peduncle) through to the cerebellar nuclei, globosus, fastigii and emboliformis, and then to the cortex of both sides. The fibres of the *superior* canals apparently have an entirely different course, going directly to the posterior longitudinal bundle, probably entering the cerebellum through its middle peduncle. Another group of fibers undoubtedly goes to the spinal cord.

We have come to a definite conclusion that all pointing reactions are primarily *cerebral* and not cerebellar, as Barany states. When one of ourselves is tested and for example with erect head is turned to the right, he has on stopping a subjective sensation of turning to the left. For this reason he feels that he is leaving the finger of the examiner behind him and points to the right, where he conceives the finger now to be. He deliberately points to the right unless by a non-automatic, calculated correction he overcomes the misconception of the position of the finger. If after the usual past-pointing, the examiner's finger is moved out to meet that of the examined, who then repeats the test, he past-points almost as much farther to the right, and would continue perhaps to the completion of an entire circle in his chase of the seemingly moving finger but for the limitations enforced by his seating. Yet if rotated and asked to make the same movements of the arm without any finger to touch, he maintains it in the same body-plane with little or no deviation, since he conceives this plane to rotate with his seemingly revolving body. There is, therefore, no cerebellar or automatic drawing of the arm to one side; the past-pointing is purely cerebral. Yet we have it achieved through the synergising action of the cerebellum: with the cerebral mandate is associated the cerebellar influence, the muscle-sense, arthrodial sense, tactile, auditory and visual impressions and memory. In fact the pointing reactions are dependent on so many things that it is necessary to assume a complicated arc to be able to explain them.

The movement, therefore, is initiated by the cerebrum, and whatever deviation occurs is because the ear-stimulus has given by artificial means a false conception of the position in space. If we postulate that the sense of direction of the right arm is determined by a stimulus going from the ear to that particular center of the cerebellar cortex governing the right arm, and from there through the superior peduncle of the cerebellum to the cerebral cortex, and similarly an entirely separate tract of fibres for the left arm center, we then can explain why any disturbance along the line of the right arm tract will cause a lack of past-pointing of that arm alone, whereas all other parts would past-point because their circuits are intact.

In conclusion, we can claim that by these valuable ear-tests we can usually differentiate lesions of the labyrinth from those of the cerebellum; we can always tell when the VIII Nerve is diseased; we can say positively also, whether or not the posterior longitudinal bundle is affected; and we can detect a lesion of the cerebellum but cannot always locate it.

We offer here two contributions that so far as we know are new:—

1. The subjective circuit. We postulate this circuit *through the cerebellum* to the higher centers and in proof cite:—(a) two patients who had perfect nystagmic reaction but no past-pointing and no vertigo; (b) several cases of spontaneous nystagmus with no vertigo; (c) in most cases of cerebellar lesion dizziness is either absent or subnormal; (d) a patient with no nystagmic reaction and yet normal past-pointing and vertigo. As the two latter reactions seem to go together, it may be concluded, at least for the time being, that the impulses which are concerned with the vertigo pass through the cerebellum. Also, arguing backwards, if no vertigo is obtained it may prove that there is a lesion of the cerebellum.

2. Our second postulate is that the fibers from the different semicircular canals have entirely separate tracts of their own; that there is one

Nystagmus
Cerebellar and } tract for the horizontal canal and an en-
Subjective

tirely different set of the three tracts for the superior canal. This we have proved in 11 cases by means of the following phenomena. In all of these there was little reason to doubt that the region of the posterior longitudinal bundle was implicated. In each of these cases douching of the superior canal (that is with the head upright) produced neither nystagmus, past-pointing nor vertigo; whereas, on stimulation of the horizontal canal (with the head back 90°) all the reactions came through promptly—nystagmus, past-pointing and vertigo. It is therefore evident that there is a complete central differentiation for the different canals of all three tracts, remembering that in none of these cases was there a peripheral lesion. Autopsy or other findings in some of these cases with no involvement of the horizontal canal fibers of either side, yet with no passing of the stimulus by the superior canal fibers, showed that in the medulla and pons the fibers for the superior canals are mesially located, whereas the horizontal canal tracts are external.

Further details might be given as showing the possibilities of scientific research in these lines—namely, the finding and defining of encephalic tracts by means of these ear-tests. Furthermore it is not improbable that we shall be able to locate in the trunk of the VIII Nerve more definitely than we have yet done, the relative positions of these bundles of fibers, as has been partly done with much value in the Optic Nerve. Just as the older anatomists recognized that the "Portio molis" and "Portio dura" could be wholly differentiated into the Acoustic and the Facial nerves of different origin and function, and as later research is separating the Cochleal from the Vestibular and Intermediary Nerves, so it is our part more minutely to differentiate these trunks into their components and by tracing their encephalic course to

improve our diagnostic possibilities, with further practical refinements in localization. To this good task we invite the aid of all our colleagues.

The paper was then illustrated by two series of moving pictures.

I. Series of normal reactions.

1. Examination for spontaneous nystagmus. Having patient look to the right, left, up and down.
2. The use of dark glasses.
3. The "Blick-fixator" of Barany.
4. Turning patient to the right with head upright, produces a horizontal nystagmus to the left.
5. Turning patient to the left with head upright, produces a horizontal nystagmus to the right.
6. Turning patient to the right with head 90° forward, produces rotary nystagmus to the left.
7. Turning patient to the left with head 90° forward, produces rotary nystagmus to the right.
8. Turning patient to the left with head inclined toward the right shoulder, produces vertical nystagmus upwards.
9. Douching water 68° into right ear produces rotary nystagmus to the left.
10. Douching water 68° into left ear produces rotary nystagmus to the right.
11. Pointing reactions in full without stimulation, i. e., spontaneous, including pointing of the shoulder from above, shoulder from below, shoulder from side, wrist, elbow, hip, neck and torso.
12. Turning patient to right produces past-pointing of both arms to the right.
13. Turning patient to the left produces past-pointing of both arms to the left.
14. Douching the right ear produces past-pointing of both arms to the right.
15. Douching the left ear produces past-pointing of both arms to the left.
16. Turning patient to the left with head 90° forward produces the marked falling reaction to the left.

II. Cases *deviating* from the normal. Series of *abnormal* reactions in cases of

1. Fistula symptom; air pressure in ear causes violent nystagmus.
2. Lesions of the labyrinth.
3. Lesions of the VIII Nerve.
4. Sarcoma of the cerebellum. (Removed by Dr. C. H. Frazier. Recovery, patient continues well after 2½ years.)
5. Other cerebellar lesions.
6. Multiple sclerosis.
7. Cyst of the right cerebello-pontine angle. Diagnosed by these ear tests and confirmed by autopsy.

Discussion.

Dr. J. Gordon Wilson, Chicago: The demonstration given by Drs. Randall and Jones was most interesting. It well showed the possibilities of the kinematograph. In the little experience Dr. Wilson has had he has found it of great use to record and study pathological conditions.

The question of pointing which has been so well brought out is very much in evidence just now. It is very essential that the observer should have a clear idea of what pointing implies. It is well to remember that pointing is a cerebral act and not a cerebellar. Further, when a patient points with the eyes shut he depends upon memory, a cerebral function, to guide him to the object. Dr. Wilson's hypothesis with regard to the relation of pointing errors during labyrinthine stimulation is different from that of Dr. Randall. Dr. Wilson believed that during the time the pointing error was present the patient had the subjection sensation of rotation, and his memory suggested the position of object. Talking in terms of the nystagmus he felt himself going in the direction of the quick phase and so he located the object and pointed in the direction of the slow phase. Pointing to a part of his own body was a different proposition. Here he depended on the kinasthetic senses and the pointing was usually accurate.

Dr. E. B. Dench, New York City, had been doing a good deal of this work at the Neurological Institute for the last few years. The examinations of the field in the region of the eighth nerve had been of great use to the neurologists. These graphic records, as they appear, are very important, enabling one to reach a more accurate diagnosis than would be possible otherwise.

Dr. Randall, in closing the discussion, wished particularly to give due credit to Dr. Jones, and to say that in establishing a semi-independent department of neuro-otology at the University of Pennsylvania, Dr. Weisenburg had encouraged

him to such a step. He thought the work thus initiated would lead a long way. He differed from time to time from the neurologists, despite the fact that they constitute a group of neurologists unsurpassed anywhere; and it was very significant when it was possible, by such methods, to conflict with the findings of such men, and to establish the correctness of the neuro-otological findings, in opposition to those of the neurologists, as he and his assistants had been able to do time and time again. It was probable that a totally different group of findings would be obtained from the tests of the horizontal and the superior semi-circular canals. At any rate only a beginning had been made in this field of work.

Dr. John B. Rae, New York City, called attention to the valuable services of Dr. W. S. Renner and Dr. A. Sharp of Buffalo, which had been rendered in obtaining the use of a moving picture machine. On motion duly seconded and carried, the thanks of the Society were extended to these gentlemen.

On motion duly seconded and carried, the thanks of the Society were also extended to Dr. B. Alexander Randall and Dr. Isaac H. Jones of Philadelphia.

THE CHORDA-TYMPANI NERVE IN OTOLOGY.

By CHARLES E. PERKINS, M. D., New York, N. Y.

My attention has been called to the consideration of the Chorda Tympani nerve and the part it plays in otology by the following cases:

1. A case reported in full before the New York Academy of Medicine in which during the radical operation there were repeated facial contractions. There was a large chorda tympani, the twitching being due to mechanical irritation of the facial transmitted through the chorda.

2. A case of ossiculectomy personally communicated by Dr. Dench in which the chorda was exceptionally large and strong so that traction upon it caused disturbance of the facial nerve with paralysis.

3. A case in which doubt arose as to whether a facial paralysis coming on immediately after a mastoid operation was due to severence of the nerve at the tip or to some condition higher up. In such a case an early test of taste sense if properly performed would settle the problem.

The chorda tympani is really an external branch of the facial. At birth it leaves the facial immediately as it emerges from the stylomastoid foramen and passes beneath the periosteum on the surface of the mastoid until it reaches the posterior end of the tympanic ring beneath which it passes through a short canal, the iter chordae posterious which will become lengthened and more deeply placed in the further development of the bone. The further course of the nerve is well known. If one holds the adult bone so that the stylomastoid foramen is illuminated the fallopian end of the posterior iter can readily be seen and a bristle passed through it.

In formulating a plan of procedure to test the sense of taste and thus ascertain the condition of the chorda tympani one finds very little aid in English literature but several

German writers have gone quite thoroughly into the subject. Schultz (1) Kander (2). The threshold stimulus varies on different parts of the gustatory surface. Thus a weaker solution of sugar will be interpreted as sweet on the anterior surface of the tongue than if applied to the posterior part or the soft palate or fauces and the converse is true of Quinia Sulphate in eliciting the sensation of bitter, so also acid and saline have their areas of minimum threshold stimulation. But as in these tests we are looking for absolute loss of taste on the anterior two-thirds of the tongue, we are not concerned with the threshold stimulus but use solutions sufficiently strong to elicit in a normal condition almost immediate response if applied to any part of the gustatory surface. I have used, simple syrup, 25% solution of tartaric acid, 25% solution of Sodium Chloride and saturated solution of Quinia Sulphate. Thus ascertaining the primary tastes of sweet, acid, saline and bitter. The solution is applied with a cotton applicator.

If the patient protrudes his tongue and we apply our solution to its anterior two-thirds and he is asked to state his sensation, in doing so he will spread the solution over other parts of the gustatory surface and our finding will be without value. Therefore we have a chart with words in large type—Sweet, Sour, Salty, Bitter and the patient is directed to point to the experienced sensation, the tongue meanwhile remaining in its original position, the solution is not disseminated and our experiment is of value. Between each test the mouth is rinsed and time sufficient to let the taste pass away is allowed to elapse. Children under ten and stupid persons cannot be accurately tested, they were, therefore excluded from my experiments.

Cases after the radical operation show complete loss of taste in the anterior two-thirds of the tongue on the operated side. I have made twenty tests in these cases and find this the invariable condition. In about one-third of the cases there was more or less loss of taste preception on the posterior third of the tongue, soft palate or fauces. Either loss of one or more tastes or misinterpretation of the sensation.

This is supposed to be due to involvement of the tympanic plexus. It seems very probable that this plexus would not escape destruction in the radical operation as now performed so we must conclude that since in but one-third of the cases we find this evidence of its involvement it is not invariably a gustatory path.

In ossiculectomy cases there is invariably loss of taste perception in the anterior two-thirds of the tongue on the operated side. This we would naturally expect as the nerve lies between the malleous and incus and would necessarily be ruptured during their removal.

In chronic middle ear suppuration as well as in the residual cases, ageusia showing involvement of the chorda is present in more than fifty per cent, sometimes we also find evidence of tympanic plexus involvement. In some of these cases the patient will retain perception for one or more tastes others being absent or he will misinterpret them showing, perhaps, that the nerve is but partially affected.

In acute middle ear processes before incision of the membrana tympani the nerve is generally intact although I found perversion of taste or loss of one of the tastes in about 20% of the cases. Thus one patient interpreted sweet as bitter, to several the salt solution seemed sour or bitter, showing partial involvement of the chorda. Ten of these cases were tested after myringotomy and showed no change. I had believed that destruction of the chorda was a common occurrence in this operation but these experiments prove that this is not the case. In order to cut the chorda the knife would have to travel along the internal tympanic wall well into Shrapnell's membrane.

This would also perhaps endanger the oval window and its contents, also the facial nerve.

In later stages of acute otitis the chorda is more frequently involved but not nearly as often as in chronic processes.

In facial paralysis neurologists make use of taste tests to aid in locating the lesion and forming a prognosis. Nuclear facial paralysis gives normal taste. Here we have also absence of involvement of the orbicularis palpebrarum, corrugator-

supercilii and anterior belly of the occipito-frontalis as their nerve supply is from the nucleus of the third, the filament to them joining the seventh just before it leaves the pons. Involvement of the nerve at the base has been reported without loss of taste by Erb (3), Ziemssen (4), Wachsmuth (5) and others; and with loss of taste by L. Bruns (6), Lehman (7) and others. If the lesion is in the fallopian canal and this of course includes all cases of otitic origin there will be ageusia. Even if involved at the stylomastoid foramen there will be sufficient swelling to impair the function of the chorda. A fact readily deduced from a consideration of the anatomical arrangement. Palsies arising from pathological conditions peripheral to the stylomastoid foramen gives absence of ageusia. This comprises a large class of cases attributed to rheumatism, taking cold, drafts of air, etc., with lesion in the neck near the foramen and together with those cases arising from the same causes with lesion at the stylomastoid foramen called Bell's palsy—a term no longer used by neurologists or others desiring to be exact. In the cases originally reported by Bell ageusia was present. Erb (9) reported twelve cases of rheumatic facial paralysis in ten of which taste sense was unimpaired. I am informed by Dr. Neustaedter who invariably makes tests of taste sense in every case of facial palsy coming into his clinic at the University and Bellevue Hospital Medical College dispensary that cases without ageusia are more certain to recover and the duration of the condition is shorter than in cases showing chordal involvement.

Bellingeri (10) in 1818 first suggested that the chorda tympani probably had some gustatory function. Claude Bernard (11) in 1843 proved that it was a gustatory nerve supplying the anterior part of the tongue, a conclusion since verified by many observers with various methods so that all agree that the taste path for the anterior two-thirds of the tongue is through the chorda tympani and facial to the geniculate ganglion, from here to the central connection a difference of opinion has existed and perhaps still does exist, as it seems almost impossible to reconcile the evidence.

The most direct way would be for the taste fibres to pass through the pars intermedia of Wrisberg to glosso-pharyngeal nucleus. This theory is apparently combatted by:

1. The occurrence of facial paralysis from basal lesions without ageusia.

2. Lesions affecting the fifth at or near the Gasserian ganglion are regularly accompanied by ageusia in the anterior two-thirds of the tongue.

3. Schiff (12) has cut the fifth near the Gasserian ganglion and produced ageusia in dogs.

4. Extirpation of the Gasserian ganglion produces loss of taste on the anterior two-thirds of the tongue on the operated side.

5. And finally a case was reported by Ferguson (13) in which a patient with chordal ageusia was found postmortem to have an exostosis in the Vidian canal with degeneration of the great superficial petrosal. These observations are supposed to prove that the chordal taste fibres reach the brain through the fifth nerve going from the geniculate ganglion through the large superficial petrosal and vidian to Meckels ganglion, thence in the second division of fifth to the brain and perhaps a part also through the small superficial petrosal to the otic ganglion and thence in the third division of the fifth to the central connection. Gowers (14) after a study of the Gasserian ganglion operations of Horsley and Ballance endorses this indirect path theory.

Cushing (15) in 1903 from a careful study of thirteen of his cases of extirpation of the Gasserian ganglion and after making repeated taste tests in each individual case arrives at the following conclusions:

1. That the perception of taste is unaffected on the posterior portion of the tongue and never permanently or completely lost on its anterior two-thirds after removal of the Gasserian ganglion.

2. That a temporary abolition or lessening of the acuity of taste may be found to exist over the anterior and anaesthetic portion of the tongue for some days after the operation.

3. That this temporary loss of function may possibly be occasioned by some interference with chorda transmission brought about by a mechanical or toxic disturbance due to degeneration of the N. lingnatis.

4. That a lesion of the trigeminal nerve may be associated with disturbance of taste over the chorda territory without the necessary inference that the nerve is a path for gustatory impulses.

5. "That the N. trigeminus in all probability does not convey taste fibres to the brain either from the anterior or posterior portion of the tongue.

I feel inclined to accept these conclusions based as they are upon such accurate and painstaking work.

As to the path for taste fibres in the tympanic plexus a number have been suggested with no prospect of accurately determining the truth of the matter.

This is readily accounted for when we consider that the plexus has three connections each one known to contain taste fibres, viz. the glosso-pharyngeal nerve, the geniculate ganglion and through the ramus communicans the chorda tympani.

BIBLIOGRAPHY.

1. Schultz. Arch. für Ohrenheilk. Vol. 79, p. 220.
2. Kander. Arch. für Ohrenheilk. Vol. 68, p. 69.
3. Erb. Deutsch Arch. für Klin. Med. 1870, Vol. 7, p. 247.
4. Ziemssen. Virchows Archiv. Bd. 13, p. 213.
5. Wachsmuth. Ueber progressive Bulbar paralyse, p. 21.
6. Bruns. Arch. für Psychiatrie 1889 Bd. 20, p. 495.
7. Lehman. Pflügers Arch. für Physiol. 1889 Supplement, p. 36.
8. Bell. Quoted by Erb. See 9.
9. Erb. Deutsch Arch. für Klin. Med. 1875. Vol. 15, p. 6.
10. Bellingeri. De Nervo Facei. 1818.
11. Claude Bernard. Journal de l'anaton, de la phys. et de la path de systemi nerveux. 1843. 1, p. 408.
12. Schiff. Quoted by Kander. See 2.
13. Ferguson. Medical News Phila. 1890. Vol. 57, p. 395.
14. Gowers. Journal of Physiology. Vol. 28, July, 1902, p. 300.
15. Cushing. John Hopkins Hosp. Bul. 1903. 14, p. 71.

Discussion.

Dr. Wells P. Eagleton, Newark, N. J., had been interested in the statement that there is nothing in English literature describing how to accurately test the sense of taste. Some years ago, having become interested in this subject, he looked through the text books, and found nothing. He then evolved a system of his own, as follows: The patient is told by the nurse that his sense of taste is to be tested on one side of the tongue first, and that if he withdraws the tongue the experiment will be lost. That he must answer the questions by signs with his head and hands. He is then instructed to protrude the tongue, a little sugar or salt is placed on moistened applicator, and then rubbed on one side of the tongue. It takes three or four seconds for this to take effect. The patient is told to keep the tongue out, and is asked, "Do you taste it?" He is told to shake his head in answer. If he did not taste it more is rubbed on, and the same question asked. If still he does not taste it, it can be stated that the sense of taste on that side is lost. The tongue is washed and the experiment repeated.

Dr. E. B. Dench, New York City, had seen a good many of these experiments carried out. Very few men were willing to take the time to work out a subject like this; Dr. Perkins' work was therefore very important.

Dr. Perkins, in closing the discussion, said that in making these tests, especially after the radical operation, the charts were so placed that the patient had the paper right in front of him. When a solution was put on the tongue on the involved side the patient looked perfectly blank but as the application was drawn forward immediately if passed to the healthy side his finger went out pointing to the experienced sensation. He thought a quicker response could be obtained by using substances in solution than by using solid substances. Slowness of the response had been considered by some to indicate a partially involved nerve.

AN IMPROVED TECHNIQUE IN THE APPLICATION OF THE THIERSCH GRAFT IN THE RADICAL OPERATION FOR CHRONIC MIDDLE EAR SUPPURATION.

By EDWARD BRADFORD DENCH, M. D., New York, N. Y.

In my experience, the application of the Thiersch graft, in cases of chronic middle-ear suppuration, operated upon by the so-called radical operation, materially shortens the period of convalescence. This method has been employed by me for the last fifteen years, with progressively better results. I admit that at first either primary or secondary grafting is not apt to meet with the success which the operator desires; at least, this was my own experience for a number of years. Persistence, however, in the plan and gradual improvement in the technique of the operator, has, in my own cases, been followed by the most gratifying results. That these results are not imaginary can be best demonstrated perhaps by the attitude which my assistants have taken regarding the use of the skin graft in cases of this character.

When I first practised this procedure, I am quite certain that members of my staff did not believe that the results obtained justified the increased time which the procedure occupied. Of late years, however, one man after another has fallen into line, until now all men working with me have adopted this primary grafting as procedure of election in cases of middle-ear suppuration which are subjected to the radical operation.

As the procedure was first carried out, it was customary to first line the entire radical cavity with a single skin graft, if this were possible; if not, to line this cavity with several smaller grafts, the grafts being held in place by pledgets of cotton, impregnated with aristol or some similar antiseptic powder; secondly, a meatal graft was used to cover the cut edges of the newly-formed external auditory meatus.

This graft was applied in one of two ways,—either by the method of Ballance, in which the graft was first applied to the posterior surface of the auricular flap, and then folded through the meatus, so as to cover the raw surface caused by enlarging the meatus, or, secondly, by the method which the writer advised for many years, namely, the suturing of the posterior wound, thus replacing the auricle, and subsequently applying the meatal graft through the enlarged external meatus, the graft being folded around the cut margin of the canal, so as to cover not only the cartilage, but also the raw surface on the internal aspect of the anterior flap which must necessarily remain as the result of separating this anterior flap from the surface of the mastoid.

Both of the above methods were open to one serious objection. This objection lay in the fact that by neither procedure was the grafted area of the middle-ear cavity continuous with the grafted area of the flap. Troublesome granulations invariably appeared just where the flap of soft tissue joined the bony surface of the mastoid. These granulations were frequently difficult to overcome, and complete dermatization of the entire radical cavity was thus delayed. While the middle-ear cavity might heal perfectly, and usually did heal perfectly in the course of two weeks, discharge would occur from the posterior superficial area for, at least, two weeks longer, owing to the fact that the denuded area had not been perfectly covered by the grafts.

In a series of cases which have been operated upon during the last six or seven months, the procedure has been modified as follows: A large graft is made to line completely the middle ear cavity. A second graft, about an inch wide and one and a half to two inches long, is then applied to the posterior margin of the bony wound, completely covering the posterior margin of the cavity, and sometimes overlapping the graft already applied to the radical cavity. This second graft is spread out posteriorly, and the portion extending into the middle-ear cavity is held in place by firm gauze packing. The portion of the graft which extends posteriorly, is then bent forward upon itself, threaded through the ex-

ternal auditory meatus and spread out so as to cover completely the cut margins of the canal. The auricle is then sutured back in place. The meatal portion of the graft is held in position by a second strip of gauze, carried through the external auditory meatus. In this way, we have a continuous cutaneous lining of the middle-ear cavity and of the margins of the meatus.

This procedure has been modified, on many occasions, by the insertion of a very large quadrilateral graft, sufficient in size not only to line the middle-ear cavity, but of sufficient length to be drawn through the meatus, in the manner already described, so that we have one continuous graft lining the entire middle-ear cavity and covering the entire cut area of soft tissues which results from the enlargement of the external auditory meatus.

The results by this improved procedure have been so satisfactory that it seemed wise to report this improved technique to the Society.

Discussion.

Dr. Charles E. Perkins, New York City, had dressed a number of these cases, and had also operated by this method. He had found the healing time much shorter, and the dressing room work much less, than by other methods. The mass of granulations usually found in the meatus did not form, and the cavity was in much better shape than in other operations.

Dr. Harris P. Mosher, Boston, had had trouble, with the Ballance method, in getting the edge of the graft into the cavity. It was interesting, therefore, to know that Dr. Dench cut his graft, using it in more than one piece. It had been his own practice for the last year to use a small graft in the middle ear cavity, and then a larger one afterward. It was difficult to make a large graft fit snugly.

Dr. Ewing W. Day, Pittsburg, Pa., added his testimony as to the efficiency of these skin flaps. He had tried the

method several years ago, but gave it up, until Dr. Dench took him in hand, in 1907 or 1908, since which time he had used nothing else. His assistant, who does all his dressings, would use nothing else. It was not as easy, however, to use this method as it might seem. He had found it advantageous to spread the graft on a piece of thin, gauze-like silk, which could then be cut off, preventing the grafts from sticking together. When the skin graft has been brought over the cut surface, and the packing has been begun, unless one exercised great care the graft would be roughed up. A drop of collodion on the graft would help to prevent this. He had not had the success in closing the eustachian tube that Dr. Dench had had. Unless the tube were closed, every time the patient blew the nose or sneezed secretions would be carried up there, keeping the area always wet. He had used a long slender catheter inserted up through the Yankauer speculum, and had cauterized the mouth of the tube. He had found that this worked beautifully. In one case the cautery point broke off. He got it out, then took a silver probe, fused some silver nitrate and a little plug of chromic acid, and firmly closed the tube. He had since done that in four cases, and had succeeded in completely closing the tube.

Dr. Mosher asked Dr. Day if he had examined these closed tubes six months later. Dr. Day answered that he had, even for years finding them closed. Dr. Mosher continued that he had found it almost impossible to close the outer end of the tube. He had put in a plug of cartilage and had found that it stayed there, the skin graft growing over it.

Dr. Dench, in closing the discussion, thought it all right to anchor the skin graft with collodion, as had been suggested, but he had not found it necessary. The important thing was to keep the graft straightened out; it should not be allowed to roll. When the graft was placed it was held in position by the assistant with a director or pair of forceps until the packing was completed. The only objection to the collodion was that it anchors the flap permanently. Some-

times it was necessary to shift the position of the graft, which could not be done if it was held firmly. Formerly he thought the graft should be anchored at the bony margin by a stitch, but afterwards found this unnecessary. He had closed the eustachian tube in one case by taking a bone graft from the mastoid. He would try Dr. Day's method.

TREATMENT OF CHRONIC SUPPURATIVE OTITIS MEDIA, WITH REPORT OF CASES.

By ERNEST DE WOLFE WALES, M. D., Indianapolis, Ind

Before taking up the treatment I will give an outline of the pathology of chronic otitis media. There is no sharp line between acute and chronic suppurative processes; the acute process tends to get well of its own accord, provided there is good drainage. If drainage is not free the resulting pressure causes further destruction and a tendency to chronicity. This chronic condition is kept up by the irritation of pus, and products of desquamation, by the growth of granulations or polyps. Even the normal mucosa becomes hyperplastic through round-cell infiltration and fibrous growth. The granulations may adhere to other granulations, further obstructing drainage, and if pressure further destruction of mucosa or bone. The bacterium, its kind and virulency, whether a mono or poly bacterial infection, as well as the resistance of the individual, all play a part in determining the outcome. Through the perforation in the drum membrane, especially if it is marginal or in Shrapnell's membrane, the epidermis of the canal grows in. This epidermis may grow over healthy bone, and is often Nature's method of cure; but if it covers over deceased bone bathed in pus, the epithelium is greatly stimulated in growth and forms false cholesteatomata. By growth these cholesteatomata increase in size and may cause pressure necrosis. After a time these cholesteatoma, through acute exacerbation, may undergo putrefaction and break down. At the same time the bone may be undergoing caries and necrosis while these changes are taking place in the mucosa or the bone may become sclerosed and fill up the mastoid cavity with compact bone. Again the bone may undergo rarefaction. Sclerosis and rarefaction of the bone may occur at the same time in different parts of the middle ear. All

these processes are Nature's attempts to bring about a cure by walling off or breaking through and establishing drainage.

The treatment of chronic otitis media may be divided into operative and conservative treatment. All complications of chronic suppurative otitis media are operative. Most cases of cholesteatomata are operative. Only the conservative treatment will be discussed in this paper. The method about to be described is not a cure-all, because after all ones common sense has to come into play to meet a multitude of conditions, but in the last ten years I have found the following treatment most satisfactory.

The ear is syringed with warm sterile water. This is necessary to get a clear picture and make a diagnosis of the condition, especially in neglected cases. Then the middle ear and canal is carefully dried with cotton and crusts are teased off with aural probe or foreign body hook. The naso-pharynx is examined and treated if necessary; the eustachian tube is blown out with catheter and the middle ear is again dried. Hearing tests are now made with tuning forks and voice. The status of the labyrinth is ascertained. The aural probe is now used and all parts examined for bare bone, granulations, products of desquamation, polypi or cholesteatomata. Then the patient lies down or rests the head on the hand, and a saturated solution of 95% boric acid alcohol is poured into the canal and allowed to remain fifteen minutes. The ear is then dried and the patient given a bottle of one ounce of boric acid alcohol with instructions to dry the ear carefully with cotton before using the drops, and under no circumstances to allow any wash water or any solution containing water to enter the ear canal. The patient is cautioned not to swim or dive. The treatment is repeated at home morning and night. It is better to give the patient the ear drops rather than have him buy them himself, so that he will come back even if he thinks he does not need treatment. The patient is impressed with constant and persistent treatment. If the drops cause too much pain or bleeding, the treatment may be stopped

for a few days and the ear dried with cotton. Soon the pain caused by the drops passes away and the patient is not caused discomfort. At first the drops tend to increase the discharge, but after a time the odor disappears and the discharge stops. When the perforation in the drum membrane is small the canal may be filled with alcohol and the tragus pressed down on the fluid, or a cotton plunger may be made and forced downward in the canal. When this procedure causes dizziness it must not be followed, but instead one may inject the saturated solution of boric acid alcohol with the middle-ear syringe. After a time absolute alcohol is used, especially if there is much hyperplastic tissue.

In the fifty cases thus treated from 1907 to 1913, but one case came to operation and that case was one in which the labyrinth was involved. Many of these cases had been advised to undergo the radical operation but had refused. They had all been treated by syringing with watery solutions. The duration of the aural discharge varied from six months to fifty-three years; the average duration was about fourteen years. Odor was present in about twenty-five cases. Evidence of cholesteatomata was present in five cases. Of the known causes of the discharge, three cases began after diphtheria, four after scarlet fever, two after typhoid, one after measles. The remainder could not give the cause. Improvement of hearing was noted in nearly all cases. In two cases polypi were removed by snare.

The effects of watery solutions in chronic suppurative otitis media is to swell polypoid mucous tissue. We know how polypoid tissue in the nose swells and patients complain in damp weather. Watery solutions macerate desquamation products and tend to break down scar tissue. Masses of cholesteatoma swell and cause terrific pain, due to the absorption of water. So in general, watery solutions hinder drainage by increasing swelling, thus making matters worse.

The effects of saturated boric acid alcohol solution is to shrink up granulation tissue and hyperplastic mucosa, thus improving drainage, and each day of treatment the solution

is enabled to reach deeper parts. After the granulations disappear the parts become covered with squamous epithelium. Alcohol hardens and prevents maceration of poorly nourished epithelium and scar tissue. Bacteria are killed, bone heals, and odor disappears, probably due to the action of the boric acid. Cholesteatomata shrinks up and comes away.

A word about wicks, because they are so commonly used in acute and chronic suppurative otitis media. Wicks act as obstructions and dam back discharges, besides causing irritation, thereby increasing the growth of granulations. It would be just as sensible to put tile drains in a river bed to make it flow better. What better drainage tube can one have than the external auditory canal?

Many cases, after radical operation, keep up discharging because watery solutions are used to cleanse them. Water macerates the delicate and poorly nourished epithelium, keeping up a chronic eczema. Treatment by boric acid alcohol quickly cures these cases just as it does chronic eczema of the canal. Two cases about to have radical operations performed were cases of chronic eczema and had normal hearing. One treatment with home treatment and they were well.

Even after the chronic suppurative ear cavity becomes apparently dry and epithelium is pale and smooth, the saturated solution of boric acid alcohol should be used a few weeks more to be sure the deeper parts of the middle ear cavity are healed. In three cases recurrence of suppuration started after colds. These quickly recovered after applying argyrol to the nasopharynx and boric acid alcohol to the middle ear.

These fifty cases all showed improvement in general health. The disappearance of odor is a great relief to the patient. The odor was the cause of seeking treatment in many cases, bleeding in a few. One case with thick masses of hyperplastic mucosa filling up the middle ear had refused radical mastoid operation, got well at the end of two years' treatment, using absolute alcohol saturated in boric acid.

This is long treatment, but the ear has been dry for five years. Most of these cases received from two to six office treatments.

The important point is that water does more harm than good, and prevents chronic suppurative otitis media from recovery. Although some cases where there is little destruction of the mucous membrane will get well in spite of water, just as some cases will get well without any treatment. As a rule, cases in which there has been great destruction of mucous membrane and bone, will not get well under the macerating effect of water or watery solutions. Alexander says in about 80 per cent. of all cases of chronic osteitis of the middle ear, suppuration defies all conservative treatment. (Diseases of Children. English translation) Bönninghaus in his "Lehrbuch der Ohrenheilkunde" says that cleansing of a chronic suppurative otitis media is only possible through syringing, and states that the so-called dry treatment is a fiasco.

These fifty cases have been dry from two to eight years, and forty-nine got well by the hydroscopic action of the alcohol treatment. It is not the hydroscopic action of alcohol on the syringe water already put in the middle ear that is wanted, but the hydroscopic action of alcohol on the pathologic tissue in the middle ear. This is the important factor. This treatment has been carried out on many cases in the clinic, but I have not been able to follow them as I have my private cases. Although there is nothing new in this method of treatment yet it differs from all text books on the treatment of chronic suppurative otitis media in that no watery solutions are used.

Discussion.

Dr. John B. Rae, New York City, suggested in addition to the rational treatment outlined by Dr. Wales, the use of suction. He had been using it in chronic cases for some time and had found it a satisfactory adjunct. He used for the purpose a silver eustachian catheter from which the ring had been cut off and which was straightened out.

Dr. J. A. Stucky, Lexington, Ky., thought wicks of gauze as drainage inserted into the canal do more harm than good. The sooner one got away from cotton and gauze drainage the better for the patient. The suction method for cleaning out the ear, mentioned by Dr. Rae, was excellent. When he used a solution for cleansing the ear which he did in the exceptional case rather than as a general rule, he employed alcohol and boric acid, and not more than twice a day.

Dr. John B. Shapleigh, St. Louis, had used the suction apparatus with a great deal of success. He had used alcohol with boracic acid, but had found no advantage in the addition of the acid.

Dr. Sargent F. Snow, Syracuse, N. Y., had used alcohol with boracic acid in some cases, but of late had been using chromic acid, sparingly applied on a bent probe. He removed granulations with a curet, then applied a ninety per cent. solution of chromic acid. This would have to be repeated several times until the bone was reached. It seemed to have an action on healthy bone. He cited a case in which he had been treating the ear for two months by this method. The patient, a girl of fourteen, had a profuse discharge and considerable granulation tissue. He had considered doing a mastoid operation, but decided to try the conservative treatment first. He curetted down the granulations, cleaned the ear with a 1-500 bichlorid solution, and then used the chromic acid. The ear had become perfectly dry under this treatment as have at least fifty others with chronic otorrhea and varying mastoid involvements.

Dr. George L. Richards, Fall River, Mass., considered it dangerous to the patient to attempt to drain through a small opening; it was better to treat the case by the open method and save the patient. In the presence of a small perforation, with a relatively small amount of drainage, cure could not be effected by any conservative method of treatment.

Dr. E. B. Dench, New York City, agreed with Dr. Richards, that the radical operation is the safe method in these cases. He cited several cases illustrative of this contention. If, however, the affected ear is the only one the patient hears with the matter should be put squarely and plainly before the patient. Sometimes, with a small perforation through Shrapnell's membrane, it was possible to effect a cure by touching the granulations with chromic acid, as in a case which he cited. The promulgation of the view that a certain number of cases can be cured by such measures was calculated to do harm.

Dr. Wells P. Eagleton, Newark, N. J., had only recently come to appreciate the importance of watery solutions in the treatment of these cases. A man who had been under his care recently, after having been treated by otologists in Newark and Boston, went to Vienna, where he was treated twice a day for two months. The discharge ceased for several years, then returned, when he consulted the speaker. It was found that the Vienna otologists had treated him with alcohol and water, which the patient was instructed to drop in the ear every three hours. He believed that syringing the ear with water did more harm than good. It was surprising, judging from conditions so often found upon mastoid operation, that more people do not die from suppurating ears, and that more do not have intracerebral complications.

Dr. John B. Rae, New York City, continuing the discussion, thought the use of chromic acid and other caustics extremely dangerous. He had observed two cases occurring within ten days of each other in service in the Manhattan Eye, Ear and Throat Hospital which tended to prove this. These cases were in the hands of different colleagues and neither colleague knew of the case of the other.

One case of chronic suppuration with granulation showed the hearing to be completely destroyed and the vestibular apparatus not functionating. The application of a bead of silver nitrate to the granulation was promptly followed by

facial paralysis. On this account a provisional diagnosis, even in presence of the dead labyrinth, of fistula involving both the horizontal canal and the aqueduct was made. This was substantially sustained on operation.

The second patient, also a case of chronic suppuration was under immediate observation suffering from vertigo, nausea, vomiting and unstable equilibrium. There was considerable hearing and the fistula test was positive. In this case an application of a saturated solution of silver nitrate was made. Facial paralysis quickly followed. On radical operation the fistula was found.

In the first case very little improvement has so far been observed in the paralysis; while in case number two, recovery has been prompt and complete.

Dr. George F. Fiske, Chicago, had had many cases in which he had employed alcohol and boracic acid successfully. Dr. Loewenberg of Paris, had employed 5 to 6 grams of absolute alcohol in 6 grams of boracic acid. He thought alcohol did good, and that boracic acid was beneficial after the alcohol had evaporated.

Dr. George E. Shambaugh, Chicago, considered the conservative treatment as outlined by Dr. Wales as very important, as it is the form of treatment applicable to the great majority of cases of chronic discharging ears. It is only in cases where the danger of a serious complication is imminent that the conservative treatment should be discarded for the radical operation. Very little difficulty is experienced, as a rule, in the recognition of the latter type of cases. In the first place it is only in the cases where the disease is actually invading the temporal bone that a complication is to be expected: that is in cases of cholesteatoma or of caries. The diagnosis of a cholesteatoma or of disease invading the bone can, as a rule be readily made by a careful examination of the type of perforation, the character of the odor, and the washings from the ear. As a matter of fact, not even in the presence of a marginal perforation in the upper posterior

quadrant or in Schrapnell's membrane, or even of a cholesteatoma, is the resort to the radical mastoid always indicated. Other clinical factors should be considered before discarding the conservative treatment. These are especially, the character and amount of discharge, the character of the odor, and the presence or absence of pain. Doubtful cases should be advised to return from time to time, since a relatively harmless case may at any time develop more active symptoms which would place it in the class that should be subjected to the radical mastoid.

It is true that one cannot promise a complete cessation of discharge by the radical mastoid operation, but it should not be lost sight of that the operation is not primarily indicated to stop a discharge so much as to relieve the patient of a condition fraught with considerable risk of a serious complication developing. The radical mastoid removes this risk, when properly done, even if it fails to put an end completely to the discharge, for example, from a patulent tubal orifice.

Dr. James F. McKernon, New York City, thought Dr. Wales treated the type of cases which he described just as all treat them, and that in the cases in which others would find the indications for the radical mastoid operation Dr. Wales would perform the radical operation. The case cited by Dr. Richards was an extreme case, in which conservative treatment would have no effect.

Dr. Charles W. Richardson, Washington, said no particular line of treatment would bring about favorable results in all cases and each case must be treated individually. He emphasized that important point that a case might cease to suppurate under one or another form of treatment, and yet, one or two years later, might begin to suppurate again. These were arrested and not cured cases.

Dr. Wales, in closing the discussion, said he had never used caustics. He reiterated the fact that the cases which he had in mind were non-operative cases. In many of them he advocated radical operation, but the patients refused, so

he went on with the conservative treatment. The ears had been dry for from two to ten years. They might yet come to suppuration, he could not tell. The treatment without watery solutions seemed to be the most satisfactory of any method he had yet tried in this class of cases.

SOMNOFORM AS AN ANESTHETIC.

By T. H. HALSTED, M. D., Syracuse, N. Y.

I have used somnoform as an anesthetic for several years, seven or eight, and estimate that it has been given for me in my operative work about four thousand times. During the first year or two after beginning its use, it was employed only occasionally and with considerable fear and anxiety because of the fact that it was a comparatively new anesthetic, and my colleagues in other cities to whom I spoke of it with increasing enthusiasm, warned me of its dangers. Nearly every one had heard of a death here and there, but on being pinned down to facts, no one of them knew definitely of a fatality that could be positively attributed to somnoform. It usually turned out that the anesthetic employed was ethyl chloride, which it is true is a large constituent of somnoform, but ethyl chloride and somnoform are not the same, the disadvantages and dangers of ethyl chloride being eliminated largely, if not entirely by the combination of ethyl chloride with bromide of ethyl and methyl chloride, which combined in certain proportions, form what is known as somnoform. If not the first, I was one of the earliest surgeons in my city to employ it, either as the only anesthetic in a short case or as a preliminary to ether in a prolonged operation. Very soon other surgeons in other branches of surgery began using it, so that today it is the usual anesthetic employed in all the general Hospitals of Syracuse as a matter of routine, as a preliminary to ether, or in short operations requiring but a minute or two, as the sole anesthetic in the case.

Somnoform is one of the most recent of the general anesthetics. While its component parts are old, the particular combination of ethyl chloride, methyl chloride and ethyl bromide, forming the combination to which was given the name somnoform, by its discoverer, Dr. G. Rolland, is new.

Dr. Rolland held the chair of Anesthesia in the Bordeaux School of Dentistry, and in 1899, after experimenting to find an anesthetic to replace nitrous oxide, discovered that somnoform answered this purpose, in that it was safe, did not require a cumbersome apparatus, provided a longer anesthesia, acted quickly and was followed by a quick revival of the patient to consciousness. He maintained that an ideal anesthetic was one that "would enter into, sojourn in, and make its exit from the body in the same manner that oxygen does; that the tension of the anesthetic agent should be greater than that of oxygen, in order that it might take the place of oxygen in the lung alveoli, and that as the degree of volatility of a gas determines its pressure, the more volatile a gas the more easily it can be absorbed, and consequently the more easily it can be made to take the place of oxygen."

Somnoform evaporates at 0 C, a fact which fulfills the objects of his definition.

The agents which were best suited to produce such an anesthetic were ethyl chloride, methyl chloride and bromide of ethyl.

Ethyl chloride, however, was found not to be volatile enough, and had a high death rate.

Bromide of ethyl was less volatile than ethyl chloride and very unstable, but in experienced hands was absolutely safe but suited only for very short operations.

Methyl chloride was found to be more volatile than either of the other two.

After experimentation, it was found that a combination of the three, in the proportion of ethyl chloride 60 parts, methyl chloride 35 parts, and bromide of ethyl 5 parts, would induce a quick anesthesia due to the presence of the methyl chloride, and a continuation of the anesthesia beyond the limits of the methyl chloride, by the presence of ethyl chloride and of ethyl bromide, which is the least volatile of the three. To this combination, Rolland gave the name of Somnoform, and under this formula it has been used several million times, perhaps most largely by dentists, and with an almost

infinitesimal death rate. Recently, because of the discovery that the excess of methyl chloride caused, after a time, a decomposition of the solution, the formula was changed to the one now used, viz. ethyl chloride 83 parts, methyl chloride 16 parts and ethyl bromide 1 part. This is the formula now employed, and is the same as that of Brugg's Mixture, an apparently identical anesthetic going under a different name although Brugg's uses the original as well as the later formula. Somnoform is made in France, Brugg's Mixture in Switzerland.

Somnoform is put up in 3 c. c. and 5 c.c. capsules, and in 60 gram vials. It is advisable to use the capsules because they are more convenient and because of the extreme volatility. The vials are not as reliable unless the contents are used up within one day on successive cases. I have only seen the capsules used.

Somnoform induces complete anesthesia in from 25 to 60, at the outside 90 seconds, and the anesthesia continues from 1 to 5 minutes, usually about two minutes, so that it is adaptable only for operations of short duration, or, a great field for its usefulness, as a preliminary to ether, relieving the struggle and the disagreeable feature of the first stage, and saving three to ten minutes in time over the use of straight ether. Somnoform is rarely followed by vomiting, or other disagreeable after effects, the patient being completely out in from 3 to 5 minutes, occasionally going on into a quiet and peaceful sleep.

The patient should be prepared as for any other general anesthetic, clothes removed or loosened, the stomach empty though this does not seem so necessary as for ether or chloroform. It does not appear to be a heart depressant, failure or respiration preceding by several minutes cardiac failure, in bad cases. Artificial respiration has always, or nearly always, restored the patient. In the beginning of the anesthesia, the pulse is rapid, due probably to apprehension, soon slowing down to nearly normal. Dr. Dudley Bruxton says there is no change in the leucocyte count, or in the hemoglobin before and after somnoform. No change in the urine,

Blood pressure is first increased, then drops slightly. The chief action is said to be first on the cerebellum, later on the cerebrum.

Before discussing the question of death rate, it might be said, as has often been said before, that in no branch of medical statistics, are statistics so unreliable as in those relating to anesthetics. There is a deep seated desire on the part of every surgeon or anesthetist, to hush up a fatality caused by an anesthetic. Dr. J. J. Buettner, lecturer on anesthetics in the Syracuse Medical College, to whom I am indebted for much of the matter contained in this paper, made an investigation of the use of somnoform in the hospitals of the city of Syracuse, and found that it had been used in these hospitals 7 or 8 years, about 15,000 times without a fatality. In addition to this it has been used probably as many times more by dentists and surgeons in their private practices outside the Hospitals, also without a fatality so far as he could discover. Dr. Rolland, in a report on the use of somnoform during the last ten years, reports 2,000,000 administrations at least, without a single fatality. Dr. W. H. DeFord, whose experience with somnoform has perhaps been greater than that of any other person outside of the discoverer, states in his book on "Lectures in General Anesthetics in Dentistry" that the death rate of ethyl chloride is 1 in 12,000, Ethyl bromide 1 in 5,000 while that of somnoform is but 6 in 2,000,000. Notwithstanding this seemingly remarkably low death rate, there is an opinion among many laryngologists and otologists of my acquaintance, that somnoform is a very dangerous anesthetic. Having heard that a somnoform death had occurred in Buffalo, an investigation revealed that this drug had not been used in the case, but that ethyl chloride had. In another rumored case in Utica, a letter from the chief of the Hospital states that somnoform had not been employed in the case at all. While this paper was being prepared, a child requiring an emergency abdominal operation was brought to one of the Syracuse Hospitals where somnoform is used as a routine as a preliminary to ether. The surgeon asked that somnoform be administered as usual, but the

anesthetist, for some reason, preferred not to, and it was not used. The child was given straight ether and died on the table from the anesthetic. A post mortem revealed an enlarged thymus, and the status lymphaticus. Had somnoform been used, the death would doubtless have been charged to it. This merely emphasizes the difficulties that surround the determination of the real cause of death of many of the fatalities attributed to the anesthetic.

Too much stress cannot be laid upon the importance of the manner of administering an anesthetic, whether it be somnoform, nitrous oxide, chloroform or ether. The ultimate result of any anesthesia depends probably more upon the personal equation of the administrator than it does upon the effect of the drug itself.

Dr. Buettner, who is an expert anesthetist of large experience and most capable, furnished me the following description of his method of using somnoform. He says:

He says:

"An important consideration is to be sure of a good fit of the face piece in order to prevent any escape of anesthetic from underneath the face piece. It is therefore necessary at times in short operations in the mouth as in the extraction of teeth or removal of adenoids and tonsils to have a mouth prop in the mouth before starting the anesthetic, such prop being of a type that will allow the mask to fit the face closely. The ordinary rubber dental props are best adapted for this. If the face piece does not fit closely, it should be covered by a towel or padded with cotton to obtain air tight results. All necessary precautions should be taken with somnoform, even for a short anesthetic, as for all other anesthetics, i. e. false teeth should be removed and there should be no tight bands about neck or waist. The most satisfactory method of administration is, having placed the patient in proper position for whatever operation is to be performed, and observing absolute quiet as far as possible, the capsule of somnoform is placed in the capsule chamber and the telescoping cap applied. The somnoform valve should be closed and the air valve open. By pressure on the telescope

cap the capsule is broken and its contents expelled in gauze or lint and retained in the rubber bag. This should never be done with the inhaler on the patient's face. The face piece is now adjusted to the face and the patient advised to breathe naturally. Experience has proven this method of breathing far superior to the instruction to breathe deeply. The patient at first breathes only air. This inspires the patient with the fact that there is no choking or strangling and that he will be able to breathe freely. Then the lever controlling air and somnoform valves is turned slightly to allow some somnoform to be inhaled but not enough to induce choking or strangling.

The lever is then gradually turned until eventually all somnoform and no air is administered. One cannot follow any definite rule of administration but must be guided by the actions of each patient. If the operation is a short one the patient is ready for work as soon as first stertorous breathing is heard. If it is a long operation and the ether sequence is to be used the anesthetist will now go on with ether. It is certainly a great advantage if ether is to be used to start with somnoform as a quicker, quieter and more peaceful anesthesia is thus obtained with a great saving of ether and the avoidance of the disagreeable odor of ether which is disliked by so many patients. In comparison with nitrous oxide alone, somnoform gives a quicker and greater anesthesia with more marked relaxation and no cyanosis. In comparison with ethyl chloride alone it is safer, quicker and gives greater relaxation with less nausea and subsequent headache.

The greatest field for somnoform is in minor surgery as extraction of teeth, opening of abscesses, reduction of fractures, pelvic examinations, paracentesis, removal of adenoids, breaking up adhesions in bursitis."

In my judgment, speaking entirely from my own personal experience as an oto-laryngologist, somnoform or Brugg's Mixture, which is the same thing under another trade name, is the most efficient, safest, most rapid and most agreeable general anesthetic for such minor operations as incision of one or both eardrums, curetment of granulations in the

tympanic cavity, opening of abscesses or furuncles of external auditory canal, or any other part, incising of peritonsillar abscess when general anesthesia is desired, as perhaps the patient himself thinks it always is, replacing of recent nasal fractures, opening into the maxillary sinus in the Mickulicz operation after the section of the inferior turbinate has been removed under cocaine, breaking down of adhesions in the nasopharynx in the adult and many other less frequent short operations. These are all operations occurring frequently in the practice of each man here, and many of you doubtless employ a local anesthetic for most of them, but I submit that in no one of them does the patient believe the local anesthetic is as perfect as it is supposed to be and in no case is the anesthesia as perfect as it is when a general anesthetic is employed.

Of course, the most frequent use of all in my practice is in cases of adenoid and tonsil operation and here a few words, giving special indications are perhaps desirable. Because of the necessity of removing the mask as soon as the operation is begun, and because of the rapid recovery to consciousness as soon as the anesthetic is discontinued, say one and a half to two minutes, I never now attempt, in the case of a child or adult, to do the complete adenoid and tonsil operation under somnoform alone, because while I have often done it successfully and satisfactorily, yet I have so often found the patient conscious before the operation was entirely completed and tonsil hemorrhage arrested, that only in very exceptional cases do I attempt the complete operation with somnoform alone.

My usual practice, in the case of a child, is to have the anesthetist get the child under with somnoform and follow with ether. In the case of adults my usual practice is to remove tonsils with sharp dissection and the snare under local novocain anesthesia, and if adenoids are to be removed, to then have the patient put under somnoform when the adenoids are removed. I use the Sluder Operation in children in preference to any other method of tonsillectomy, and if tonsils alone, without adenoids, are to be removed, somno-

form is sufficient, but if the tonsils are to be dissected and removed with the snare, somnoform alone will not be sufficient in the majority of cases.

In operations to be prolonged more than two minutes, ether preceded by somnoform, is the routine.

In conclusion: —

1. Somnoform is the ideal anesthetic for both children and adults for short operations, producing profound anesthesia in from 30 to 90 seconds, allowing a period of from one and one-half to three minutes for operating. The recovery to consciousness occurs usually within three to five minutes after discontinuance of the anesthetic.

2. It is as safe as nitrous oxide anesthesia, is as rapid in its action, recovery being equally rapid, does not produce cyanosis, has no serious after effects, and because of the simplicity of the apparatus and ease of transportation, is preferable to nitrous oxide for operations of short duration.

3. The mortality is lower than that of any other general anesthetic in use, unless it may possibly be nitrous oxide gas.

4. As a preliminary to ether, somnoform has a very great field of usefulness, eliminating largely the disagreeable struggle and excitement of the first stage of ether, shortening the time by three to ten minutes, and lessening the quantity of ether used.

5. A competent anesthetist is as desirable in the administration of somnoform as in the employment of any other anesthetic.

Discussion.

Dr. Harris P. Mosher, Boston, said somnoform had appealed to him very strongly, and he had used it; but word had come to him through medical gossip that if he continued to use it he would have a death on his hands, and so he had discontinued its use. This was a disappointment to him, for he had found it very convenient to carry around. He had been waiting for someone to go over the statistics of the subject, as Dr. Halsted had done, that he might begin again

to employ it. It had been used in a hospital with which he was connected, as a preliminary to ether, but had been abandoned.

Dr. Ewing W. Day, Pittsburg, Pa., thought the personal equation the keynote to the question of anesthesia. Dr. Simpson, who, four years ago, was the anesthetist of the hospital with which he was connected, used somnoform extensively and had no difficulty with it. The next anesthetist would not use somnoform at all, but employed ethyl chlorid, with just as good results. The third anesthetist gave straight ether, with as good results as either of the other two had with the anesthetics of their choice.

Dr. Sargent F. Snow, Syracuse, N. Y., corroborated what Dr. Halsted had said concerning the use of somnoform at the Syracuse Hospital. Ten years ago, while he was still in active hospital and dispensary work, he used bromid of ethyl, which he found satisfactory in trained hands. At present, however, he preferred somnoform because it gives less rigidity of the muscles. He had found somnoform, followed by ether, very satisfactory.

Dr. Halsted, in closing the discussion, said he had brought the subject up hoping that if anyone knew of a death from somnoform it would be mentioned in the discussion. He had heard of a good many deaths, but some of these, when investigated, proved not to be due to somnoform at all. It was not necessary to have an expert anesthetist. It is the only general anesthetic that he ever gives in his office.

A MODIFIED RADICAL MASTOID OPERATION.

By J. A. STUCKY, M. D., Lexington, Ky.

The indications for the so called radical mastoid operation is interference with free drainage and ventilation of the middle ear cavity due to retained septic material in the aditus ad antrum, mastoid cells, and necrosis or caries of the ossicles and the walls and floor of the cavity. The radical operation converts into one cavity the middle ear attic, mastoid process and auditory canal, and is regarded as a major operation and one of the most important and most difficult in otology. On account of the liability of injury to the facial nerve and the vital and easily exposed portions of the labyrinth, it is approached with more trepidation and anxiety than all other operations combined in otology.

The after treatment is usually long and tedious in order to get the cavity entirely healed and lined with a vascular fibrous membrane free from irregularities and pockets. The hearing after healing has taken place is rarely ever better than before the operation and is frequently more impaired. Notwithstanding these drawbacks and criticisms this operation has probably prevented more cases of meningitis, relieved a larger number of cases of chronic foul smelling discharge from the ear than all the local, systemic and surgical treatment combined. In many cases, however, I have found that a modification of this (the Stacke-Swartze) operation accomplishes the same results without the long posterior incision, with less danger of injury to the facial nerve and labyrinth and with a much shorter and less painful after treatment. Unfortunately the operation can only be performed in a limited number of selected cases, those (a) with a large auditory canal and external opening (b) those in which there is no eburnated or sclerosed bone (c) those without lateral sinus involvement or in which there is an extra dural abscess or a very narrow aditus ad antrum (d) those in

which there is no involvement of the middle and lower portion of the mastoid process, but where the disease is confined principally to the middle ear, aditus and antrum. For these reasons a careful selection of cases is necessary, aided by the radiogram and transillumination.

In three years I have performed this operation only twenty-six times but the results have been so gratifying in this number that I think the matter of sufficient importance to present to this Society for its consideration and discussion.

In suitable cases for this operation, we usually find the middle ear cavity filled with bands of adhesions or soft polypoid granulations—The first step of the operation is to free these adhesions with small, short blade, pointed knife as advised by Dench (Disease ear pp. 488) for removal of the ossicles. As already stated in all of these cases I have operated, there was decided caries of both malleus and incus often only small piece of each being held in the middle ear or aditus by fibrous bands of adhesion—these serving to lock up the retained septic material which in time is absorbed, resulting in the varying degree of septicemia usually found in these cases. With the aid of adrenalin solution and compression, bleeding is easily controlled, and every step of the operation readily seen. Great care should be exercised in order to thoroughly remove the carious ossicles, also not to disturb the stapes or fracture its crura—the middle ear cavity should be freed of all granulations and fibrous bands of adhesions with knife and curet. When remnants of ossicles (malleous and incus) can not be found and removed with forceps and curet, the use of Dench's incus hook is a valuable aid. To this point in the operation, the technique is about the same as that described by Dench in both the first and second editions of his work already referred to in Chapter on ossiculectomy.

Especial attention is now given to the middle ear orifice of the eustachian tube with curets devised by Dench, Yankhauer and the writer's olive shaped hand drill. The orifice and part of the eustachian tube is thoroughly cleaned and left in condition to stimulate adhesive inflammation which will

result in its permanent closure. The most important step of the operation, is the removal of the over hanging tympanic ring with bone forceps devised by Dench (Fig. 1) and Hartman (Fig. 2). These forceps are small and of superior

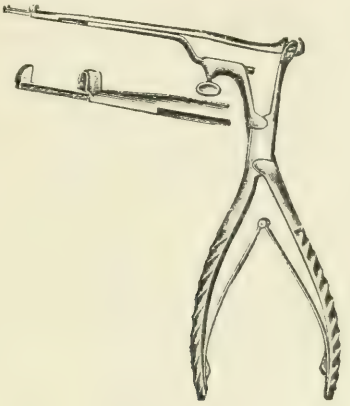


Figure 1.

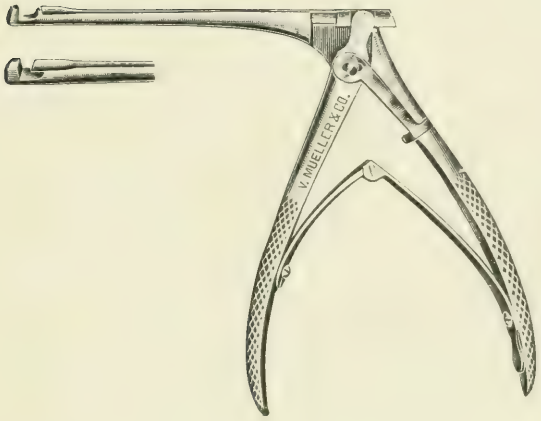


Figure 2.

strength and will easily bite off chips of bone from the over hanging bony ring—being so constructed, that when open the distal extremity of the lower blade can be easily passed up behind the tympanic ring and upon closing the instrument the chisle blade cuts away a

small chip. This chip of bone usually hangs in the grooved lower blade of the forceps and must be removed before attempting further use of the instrument. In cases, in which this operation can successfully be performed this biting off process can be continued until the aditus ad antrum, and occasionally the antrum and part of the superior posterior wall is removed.

I am convinced that many cases of chronic otitismedia, even those so far advanced that the membrana tympani and part of the ossicular chain is destroyed, the suppuration is kept up not by disease in the middle ear, but on account of caries of the ossicles and necrosis of the aditus ad antrum, which prevents ventilation and drainage of the antrum. In a large number of these cases the pathological foci is held within the aditus ad antrum and mastoid antrum by a low over hanging tympanic ring. I have been surprised at the prompt relief of some cases following removal of the remaining portions of caried ossicles and the upper margin of the tympanic ring—thus permitting of free drainage and ventilation.

Care should be used in selection of cases. (1) Where the auditory canal is too small to permit the use of speculum of sufficient size to freely admit passing of the several instruments necessary to complete the operation. (2) When the radiograph and transillumination shows a very narrow aditus ad antrum and a thick, dense, or sclerosed posterior superior wall, with the antrum located far back, the operation suggested should not be attempted. In such cases the radical mastoid (Stache-Swartze) operation alone is indicated. Little or no cureting is advised—but if used great care should be exercised that no injury is done the stapes, or the oval or round window.

After removing all the bone necessary, thoroughly cleansing and drying the middle ear cavity, the superior portion of the external membranous lining of the canal is carefully replaced and held in position by iodoform gauze. This is allowed to remain for twenty-four to forty hours and is then to be removed by moistening with hydrogen-dioxide so as

not to disturb the flap made of the external membranous canal, or to cause any bleeding as the result of rough handling. The subsequent treatment consists in gently wiping out the middle ear cavity four or six times in twenty-four hours, followed by the immediate use of boracic acid by means of a powder blower until there is no further discharge, and dermitization is complete. This usually is accomplished in from one to three weeks. No gauze or cotton drainage is used after the first or second dressing is removed. A small portion of cotton is put into the bowl of the external ear to absorb drainage and moisture, and is removed as often as soiled, a fresh pledget of cotton being applied.

I make no claim to presenting anything new, but the results in the twenty-six cases, that I have been able to use this method have been such as to warrant confidence and further trial. The method used has been more than the removal of various ossicles—this alone I am convinced would have resulted in failure in any of the cases to which I have referred, after the ossicles (or remaining portions of same) are removed, and the cavity of the middle ear thoroughly cleaned, the operation is only half completed—the overhanging tympanic wall must be removed, and the biting off of chips of bone continued until the biting forceps can no longer be made to take hold. When this is accomplished we usually have obtained free and unobstructed drainage and ventilation of the attic and antrum. No packing or gauze strips for drainage is to be used—nor any solutions dropped into the ear. Neither have found irrigation to do aught but injury in this class of cases. The use of the boric acid powder is to be continued from four to six times in the twenty-four hours, until the middle ear is dry—after this it should be used two or three times a day for a week or ten days. That there is place in Otological surgery for ossiculectomy and this modified radical operation, I am assured by the results in the twenty-six cases already referred to. In none of these has there been any return of discharge nor has there been any decrease in the

hearing. The patients are kept in bed only twenty-four hours and are discharged from the hospital in from three to five days.

Discussion.

Dr. Frederick L. Jack, Boston, said the major part of the operation is the old middle ear operation for removing the drum and ossicles—a method he had advocated in the early history of the radical operation, and which he had continued to use despite adverse criticism. He had found that in removing the bony portion, in making one cavity of the attic, he was bothered with exuberant granulations which delayed healing. His original method of clearing out the middle ear gave quicker healing. Perhaps the cases in which he operated had no trouble with the antrum, merely with the middle ear. As to closure of the Eustachian tube, he was not so sure in his own cases that the tube was closed. He was not convinced that infection always came through the Eustachian tube; he thought perhaps something was left in the bone which subsequently broke down. He employed a set of small ear curettes which he had devised for this middle ear operation. He had been surprised to find that in the radical and middle ear operations the hearing was improved following the operation. He recalled a case in point in which there was chronic suppuration, in which, Dr. Mosher, during his absence, had performed the radical operation, with improvement of the hearing. The patient was now suffering with chronic suppuration in the other ear, and wanted a radical operation with the idea of improving the hearing on that side. In none of his cases had the hearing been destroyed, and in many it was improved.

Dr. E. B. Dench, New York City, asked the essayist why he made an incision in the canal, and whether it was for the purpose of baring the bone. When he first described his technic for ossiculectomy he made a particular point of the necessity of rotating the long process of the incus from behind forwards, rather than from before backwards. He asked

Dr. Stucky if much trouble had been experienced in getting cases to dermatize. In most of his cases dermatization was not satisfactory over the promontory. The chisel-forceps had proved an excellent instrument in his hands. Neumann used a small gouge, but in a case operated by Neumann for him the operation was unsuccessful, and Dr. Hopkins subsequently performed a radical operation. He had had to do the radical operation after from five to six months to three or four years, and one could not pronounce a case cured until five or six years had passed without discharge. There was danger of facial paralysis. The worst case of facial paralysis he had had followed the use of his cutting forceps. Whether or not there was an overhanging facial ridge he did not know. In carrying this instrument backward one might go through a very thin Fallopian canal, injure the nerve and cause facial paralysis. It was important to bear this in mind. He was glad to note that all through the discussion of the radical operation many had testified to the improvement of hearing after the radical mastoid operation. The majority of otologists had always taken the ground that the hearing would be lost; he was glad to see the opinion swinging the other way. He had had an experience similar to that reported by Dr. Jack, of a patient having the hearing improved after the radical operation on one side and returning later for operation upon the other side. Unless the hearing was exceptionally good it would not be impaired by a radical operation. In the presence of normal or increased bone conduction and no interference with the upper tone limit, one could predict that the hearing would not be impaired by the radical operation unless the hearing had not been at all or only slightly impaired by the aural suppuration.

In cases where in spite of prolonged suppuration the hearing remained practically normal, it would probably be somewhat less acute after the radical operation. In many of these cases however, no impairment of hearing resulted.

Dr. Ewing W. Day, Pittsburg, Pa., thought this method might be safe in the hands of Dr. Stucky, but if generally followed there would be as many cases of facial paralysis as with the radical mastoid operation. One of his confreres in Pittsburg had employed the method, with two cases of facial paralysis resulting.

Dr. Stucky, in closing the discussion, said he did not want to convey the impression that the modified operation was to take the place of the radical, but that there was a place for it. The mistake which Dr. Rae had found beginners apt to make had caused the speaker to abandon the operation because he had lost sight of the fact that the patient was in the recumbent position, that he was beginning to bite off the bone in the wrong direction, instead of making straight for the attic. By keeping this in mind the difficulty could be obviated. The longest period during which he had been able to keep track of a patient was three years. Dermatization was complete and there was no discharge. He had been able to see all of the twenty-six cases within the last six weeks. In all of these the attic, the aditus, antrum, and middle ear were made into one cavity. The greatest danger in the operation was the too-free use of the curet, against which he had cautioned in the paper.

ACUTE SUPPURATIVE OTITIS MEDIA WITH UNUSUAL COMPLICATIONS.

By EUGENE R. LEWIS, M. D., Dubuque, Iowa.

The following extract is taken from the history sheet of a case which caused me a great deal of worry, which ended very happily, and which since has furnished profitable and interesting material for study. The patient, an eight year old boy, had had pertussis at two years, measles at six, acute anterior poliomyelitis at seven, severe bronchitis and bronchial asthma during the fall and winter previous to present illness. After two months in the South returned home much improved in health and strength, having increased from sixty to seventy-two pounds in weight.

After five days of moderate acute afebrile rhinitis, developed left otitis media acuta. Free incision early; vomiting and pronounced disturbance of static equilibrium throughout the day; nothing unusual until four days later, after very moderate activity, sudden onset of intense headache, fever, prostration, subjective equilibrial disturbance without nystagmus.

During the following three weeks temperature fluctuated between ninety-nine degrees and one hundred five and eight tenths, pulse between eighty and one hundred fifty, respirations between fourteen and forty-eight during sleep. Irritation symptoms in sensory, psychic and motor fields, particularly general muscular hypertonus, evidenced by stiffness of neck and Kernig symptom; repeated copious epistaxis, prolonged optic nerve hyper-sensitiveness, leucocytosis fluctuating between ten thousand and forty thousand, rapid emaciation—loss of weight from seventy-two pounds to fifty-two pounds; enormous excess of phosphatic elimination via the urine, which was unvaryingly of acid reaction; late in the disease slight albuminuria with granular and hyaline casts of small calibre was present; cultures made from the

pus from the middle ear at the height of the disease and blood cultures showed streptococcus of an unusual type in that the organism apparently underwent morphologic transmutation, showing in direct smears of pus as a Gram negative diplococcus, and yet showing in the cultures as a pure streptococcus.

At no time was there any sign of mastoid involvement or of acute accessory sinus empyema, though occasional nasal discharge of mucopurulent character was present throughout much of the disease.

The character of the equilibrial disturbance was not the same as that seen in labyrinthine or cerebellar disease except on the first day of the otitis media. The patient on several occasions volunteered the information that things seemed queer, "my feet feel up and my head feels down," suggesting much more that type of static equilibrial disturbance associated with disease of the temporal lobe; also suggestive of affection of this area were the tendencies shown on several occasions toward paradoxical facetiousness, or "Witzelsucht."

During the whole course of this illness the patient was on an almost exclusive milk diet, with negligible additions of crackers, toast, cocoa, and occasionally ginger ale. The appetite was very poor, tongue coated, and bowels decidedly sluggish. Repeated Widal tests negative.

Defervescence was gradual, and after normal had been reached, there was a rise of from one and a half to a half degree every day, not at regular times, for over two weeks during the steady progress of convalescence. The otitis media subsided in about ten days after the declining temperature reached normal; membrana tympani remained freely open all during the course of the disease and closed promptly on cessation of discharge. No impairment of hearing acuity six weeks after. Subsequent history of complete restoration to health and strength over a year's time.

This is the third case of its kind that I have seen; two of the three recovered completely, the third died, apparently exhausted after three weeks illness. These cases had several

characteristics in common; in all three the beginnings had all the ear-marks of the commonplace—relatively slight local symptoms of rhinitis for several days—followed by unexpected extension of the infection or at least, of inflammation, into some area contiguous to the intracranium. These extensions rather than the original inflammatory process seemed to be responsible for the subsequent gravity of the cases. Possibly by reason of anatomic anomaly, dehiscence, or unusually direct vascular connections with the intracranial tissues, more probably because of inadequate protective organization of circuminflammatory exudate, the course of what, judging from physical findings, should have been ordinary acute otitis media, became clouded by very grave symptoms of intracranial disease. In none of these cases was there at any time a physical sign of mastoid disease; in none of them was there at any time a suspicion of inadequate drainage of the tympanum.

These cases of "meningism" or "pseudo-meningitis" are more likely to develop in individuals who have had more or less profound nutritional disturbances prior to the onset of the acute underlying infection; they are more frequent in children, especially in the rachitic and in those who have shown tendencies toward infantile scurvy and purpura hemorrhagica. There seems to be ground for the belief that bio-chemic perversions of tissue metabolism constitute a predisposing cause. For example, the existence of subnormal calcium content in the tissues at the onset of the infection would explain several obscure features of the case reported. Low calcium-content of the blood causes faulty fibrinogenesis, which interferes with organization of circuminflammatory exudate, whereupon lymph- and serum-borne toxins seeping through the imperfectly formed barriers set up a non-purulent encephalitis. Oppenheim, collating necropsy findings in case reports by Kohte, Dupre, Seitz, Lepine and others, reports "no evidence of purulent inflammation in meninges and contiguous brain tissues; in some instances absolutely no departure from normal, in others edema, hyperemia, and occasionally serous exudation."

These findings at necropsy, coupled with the clinical experience that those cases which are not fatal go on to complete recovery, bear out my impression from study of these cases that, whatever the character of the encephalitis at the height of the process, it is not such as to leave recognizable changes postmortem, or as to interfere with complete restitutio ad integram in cases terminating in recovery. Yet during the course of the disease one may find not only headache, equilibrial disturbances, and mental torpor, but also delirium, general convulsions, general hypertony, and even neuritis optica. It is hardly conceivable that such an array of symptoms may arise without definite physical changes in the cells affected. The failure to find pathologic evidence of such cellular changes argues less their non-existence than it does the viability and retention of cellular integrity in spite of the existence for a time of wide departures from the normal in cellular metabolism.

In this connection the evidence of the urinary findings is particularly significant. The enormous phosphaturia observed over a considerable period of time indicates rapid katabolism in cells of high phosphorus content. In the presence of sufficient tissue-calcium, fixation of this phosphatic excess in the form of calcium salts and elimination by the intestinal tract would naturally occur, leaving only a comparatively small proportion of the excess to be disposed of by the kidneys.

Lumbar puncture as an aid to diagnosis is of questionable value except under conditions affording unusually advantageous interpretation of findings. The presence of polynuclear leucocytes in the cerebrospinal fluid indicates the probability of purulent meningitis; but similar findings are present in simple cortical abscess cerebri. And, on the other hand, should occlusion of Magendie's foramen abolish communication between subarachnoid space and ventricles during the course of diffuse purulent encephalitis, spinal fluid would show no leucocytes. The albumin content of the spinal fluid is said to be increased in

cerebral abscess and purulent encephalitis, not in non-purulent encephalitis or meningitis serosa.

Differential diagnosis between the purulence and non-purulence of acute encephalitic complications of nasopharyngeal and aural infections is extremely difficult, if not impossible. The most reliable criterion is the outcome of the case. Cases which recover are non-purulent; the nature of the others must be determined by autopsy findings.

Discussion.

Dr. E. B. Dench, New York City, thought the case looked like one of sinus thrombosis, in view of the fluctuations of temperature between 95 and 105 degrees and the positive blood culture. There was probably a clot which gave rise to systemic infection. He had reported two such cases a few years ago. When one remembered the intimate relationship between the dome of the jugular bulb, the lymph channels and the subarachnoid space, it was easy to see how a clot in the jugular bulb could cause disturbances of equilibrium where the labyrinth was not absolutely dead. Dr. Lewis' case looked to him like one with a parietal clot and spontaneous recovery.

Dr. George E. Shambaugh, Chicago, had seen the case a couple of times with Dr. Lewis and in both instances had advised against a mastoid operation. The positive blood culture was obtained just as the child began to improve. Had the positive cultures been obtained when the fever was highest it is possible that an operation would have been advised. Cases of rather high temperature, associated with an acute otitis media, are sometimes very puzzling, especially when they occur in children. The absence of any distinct symptoms of a mastoiditis by no means excludes the danger of a sinus thrombosis or of meningitis developing. One should always remember that a rather high temperature in a child does not have the same serious significance as it does in an adult. In this case the possibility of a meningitis was foremost in their minds, but the child during the entire

process had not only a suppurating ear but a purulent discharge from the nose. It seemed as likely that the temperature came from the condition in the nose as from the ear.

Dr. Emil Amberg, Detroit, Mich., called attention to the fact that there might be a central pneumonia which could not be detected by any known method now in general use. He was told that perhaps the X-ray examination might assist in the diagnosis. He had two or three such cases. In one case exhibiting meningeal symptoms, a spot was found in the right lung by ordinary means. The patient recovered without operation (reported elsewhere under the title "Meningisms in Pneumonia").

Dr. Wells P. Eagleton, Newark, N. J., considered the difference between suppurative encephalitis and non-suppurative encephalitis only a question of degree. In non-suppurative encephalitis the case did not go on to the same termination; in every case there was the possibility of brain abscess. Lumbar puncture was a valuable aid in diagnosis and also as a therapeutic agent. Any trouble in the head of an infective nature, whether suppurative or not, was apt to be shown in the chemical changes in the cerebrospinal fluid. Lumbar puncture in typhoid fever with delirium, for example, would show changes in the cerebrospinal fluid. In the large majority of cases the simple drawing off of the fluid lessened the pressure under which the cerebrospinal system was suffering. In every case of brain abscess the same thing was found—there was increase in the amount of cerebrospinal fluid, with changes in its composition. In Dr. Lewis' case the diagnosis would have been materially helped by lumbar puncture. He cited a case in which the patient had been returned to a more or less normal condition by repeated lumbar punctures alone. She probably had tuberculus meningitis, but the simple withdrawal of fluid gave a return to a more nearly normal state than she had been in for several days.

Dr. Lewis in closing the discussion, reiterated that, at the time, they had not been at all certain as to what sort of intracranial process they were dealing with, and, in the absence of more definite indications they had not subjected this child to operation. The continued satisfactory local conditions of the mastoid and middle ear in the matter of adequate drainage directed close attention to the coexistent nasal infection, concerning the importance of which as a possible infection focus there was some difference of opinion. Both aural and nasal infections underwent gradual subsidence, and intracranial symptoms disappeared along with them. The recovery was complete with restoration of normal nasal and aural conditions.

The temperature was never of a septic type. It would run for three or four days at 101°, then would rise to perhaps 104°. Without any extraneous influence it would fall to where it was before, run there for a time, then go up to 105° plus.

One symptom of the complicating intracranial condition not detailed in the paper was the incidence of sudden head pain; on several occasions the child awakened from a sound sleep, complained of agonizing pain in the left half of the head, which continued for an hour or an hour and a half, and as suddenly disappeared.

There were other causes for the leucocytosis, so that it was not attributed solely to the head conditions. At no time was there a chill or a suspicion of a chill, no sweat, no nystagmus.

The pathologist at St. Luke's Hospital, Chicago, was sent for and all blood and bacteriologic examinations were made by him.

Dr. Lewis regretted that Dr. Eagleton was not more specific in stating that lumbar puncture would have been valuable in diagnosis of this case. For the reasons given in the text, the essayist still believes that lumbar puncture offered but little toward diagnosis of this case.

REPORT OF A CASE OF BRAIN ABSCESS WITH UNUSUAL FINDINGS.

By GEORGE L. RICHARDS, M. D., Fall River, Mass.

The following case of brain abscess seems of sufficient interest to report, even though it was an unsuccessful one. I have the feeling that the statistics of brain abscess as published are better than facts really warrant, since many of us hesitate to report and put on record our unsuccessful cases.

The patient a Swede, carpenter by occupation and twenty-four years of age, was admitted to the Union Hospital in Fall River on the evening of April 14, 1915. The family history was negative, but he said he had been sick several years ago with hemorrhages of the lung but had recovered. The only thing in the immediate previous history of moment was the fact that he had two or three fingers bound up as the result of a scratch or injury in his work and on examination similar sores were found on other parts of the body. He stated that a small wound of the hand or elsewhere healed rather slowly.

Present History. Three weeks ago he had some trouble with his right ear. He consulted a physician who incised the drum. This gave relief for a short time but the pain returned, accompanied with sudden and severe headaches. A physician was called who treated him for a few days for these same symptoms. In addition there was some nausea but no vomiting. There being no improvement and his mental condition becoming somewhat sluggish he was sent to the hospital, where, owing to my absence from the city, he was first seen by another member of the staff who incised the drum with some relief. At this time there was only slight pain in the ear, and no mastoid tenderness. I saw him for the first time the morning after he came to the hospital, when the drum incision had almost entirely closed

except at one small point, and I re-incised it. He complained of no pain on pressure and there was very little discharge after the drum incision. Pulse 56 to 60, temperature 99, respiration about 24. Mental condition a bit sluggish but answered questions fairly well, although never giving a good account of his present illness. It was impossible to find out from him just when the drum was first incised or any clear account of the initial symptoms. His principal complaint was of severe headache, rather frontal in character, relieved somewhat by the ice bag and this headache never entirely ceased. He was in bed with the ice bag over his head, eyes closed, apparently stupid, but would rouse to answer any question. There was no muscular rigidity or paralysis, the reflexes were all present but slightly sluggish, and there was no nystagmus. An ophthalmoscopic examination of the right eye showed a choked disc of about three diopters.

April 16th there was only a slight discharge from the ear but several hours would elapse before the small wick placed in the external canal was wet through. An X-ray picture was taken and a diagnosis returned of infiltration in the region of the right mastoid probably extending well up into the middle fossa. The pulse dropped lower and ranged all the way from 45 to 58. He asked for the ice bag constantly and lay in a rather stuporous condition, but said he was all right and would be as well as ever in a few days if let alone. Later on, in the evening of this day the symptoms seemed to grow worse, the temperature went up to about 100, and the diagnosis of brain abscess in temporosphenoidal lobe seemed to be plainly warranted even in the absence of any paralysis, so he was made ready for operation. On entering the operating room however he refused to be operated upon, and every effort to gain his consent failed. He maintained that he was twenty-one years of age, his own boss, and that we had no right to do anything to him. If we would only let him alone he would be all right. He would fool us all yet and be all right in the morning. He was therefore returned to the ward.

April 17, the third day after admission, the pulse was 40, temperature normal, and the respiration 24. The choked disc had increased to almost five diopters, the cerebration was more sluggish, reflexes less active, and the ear discharging very little. He was still complaining of severe frontal headaches but there was no pain over the mastoid. A telegram was sent to the patient's father. He arrived the following morning and after much difficulty obtained consent for operation which was done at three P. M. the same day. He took ether rather slowly requiring three-quarters of an hour, but was easily kept under once it was attained.

The usual operation for an acute mastoid was first performed. There were a few soft cells and a drop or two of pus in the region of the antrum but no general breaking down of the mastoid cells. The antrum was rather higher and deeper than usual, a millimeter or two above the spine. No apparent cause for his symptoms having yet been found, the incision was extended upwards and forwards and the bone removed until the dura was bared. There was no apparent pulsation so with the aid of ronguer forceps the bone was cut away an inch to an inch and a quarter above the antrum. As the ronguer was being used in this region a slight flow of pus appeared on the surface. As more bone was cut away the flow of pus increased. Considerable amount of bone was now carefully removed when it was found that we were directly over a large abscess cavity, beginning a very short distance beneath the dura and extending towards the centre of the brain and anteriorly. A crucial incision was made in the dura and the brain structures incised. The abscess was so near the surface that it was not necessary to make a very deep cut. From one to two ounces of pus were evacuated. Careful examination with the probe showed a cavity without any definite walls. The bulk of the pus being evacuated rubber drainage tubes of the collapsible type, three in number, were pinned together and inserted about an inch to an inch and a half into the abscess cavity and anchored to the skin edge. The idea in using these tubes was that the drainage would occur in the

spaces between them as well as through the tube, and that they would not block up, while if the cerebral pressure increased very much they would easily be forced out, nor would they traumatize in any way. It is well known that the problem of drainage in abscess cases is a difficult one owing to the soft and easily collapsible walls of the abscess cavity. The wound was not sutured at any point. The operation lasted about one hour and at nine P. M. he seemed decidedly better, was apparently conscious, the pulse had gone up to 67 and his general condition seemed improved. April 19th he had a restless night and complained greatly of pain the following morning so the wound was dressed. The rubber tubes had been pushed out and so were replaced by dry, gauze wicks. A small amount of pus was steadily exuding. During the day the patient grew gradually worse and by evening was almost entirely unconscious. The pulse had again gone down to 48, the reflexes were quite abolished, and his only conscious remark was "Such a terrible headache." The interne reported that during the night there was a convulsion. At two o'clock the following morning, April 20th, he died.

Laboratory Findings. He came to Fall River from Paterson, N. J., and gave a history of having had chills and vomiting spells. The chills he said he had had off and on for several years. Examination of the urine and the blood showed in the blood a white count of only 6,000; Hb. 80%; Poly's 63%; large mono. 18%; small mono. 16%; eosin 2%; mast 1%. With this unusual picture in a case apparently due to a mastoid or some abscess connected with the mastoid a search was made for typhoid, tuberculosis and malarial organisms. Tuberculosis organisms were not found, the Widal reaction was negative, the malarial plasmodium were found on two examinations of the blood, and there was an enlarged spleen about two fingers breadth below the ribs. In the first specimen of unstained blood these organisms were very motile and seen very plainly. There was nothing definite or unusual discovered in the urine except almost a pure culture of colon bacilli. A spinal

puncture was made just before the operation, the patient refusing this before, also refusing blood for a Wasserman. An examination of this spinal fluid showed 343 cells to the square mm. and a culture growth of staphylococcus, and a negative Wasserman. Abderhalden was not done. Spinal fluid taken at the time of the autopsy, twelve hours after death, showed a strongly positive Wasserman and a culture growth of pneumococcus. The culture made from the pus from the abscess showed pneumococcus. Pneumococcus was also grown from the spinal fluid taken at the autopsy.

Partial autopsy made by Dr. D. J. Fennelly, Hospital Neurologist. Time twelve hours after death. Brain exposed by removing a large area of bone, vessels of pia injected throughout, more marked in right hemisphere. Tried to remove brain en mass but this was impossible on account of bogginess of substance. Removed part of cerebrum and part of cerebellum. Following incision of operation the abscess cavity was found directly beneath the opening that had been made at the operation for drainage. The cavity was found to be about two inches wide by one and one-half in depth. This had ruptured into the right ventricle and the ventricle contained a semi-liquid pus. The bones about the tip of the mastoid and the labyrinth macroscopically had an almost normal appearance. Microscopical examination was made of the brain tissue, sections being stained after the method of Nissl. Chromatolysis had taken place, the cell bodies being considerable swollen and the nucleus in many places peripherally located. Sections were also stained for intracellular fat and this was found to be present, not much fat was found in the nerve fibers.

Some points of interest are first,—Almost entire absence of tenderness over the mastoid at any time during his illness; second, extremely late abolishment of any of the reflexes in so extensive a process as the partial autopsy and operation proved; third, low leucocyte count; fourth, presence of malarial organisms; fifth, colon bacillus in the urine; sixth, pure culture of pneumococcus in the pus and the spinal fluid after death; seventh, negative Wasserman before death

and a triple positive a few hours after death: eighth, absence of a cavity wall.

Did the malarial organism alone account for the leucopenia or difference in the Wasserman reactions? How account for negative Wasserman a few hours before death and a strongly positive a few hours after death? (This fluid was divided into several tubes and all the tests were made at the same time with the same results, each test tube containing post mortem fluid was positive and each ante mortem negative). What had the colon bacillus to do with the case?

Discussion.

Dr. E. B. Dench, New York City, agreed with Dr. Richards that lumbar puncture made earlier would have settled the diagnosis. He was at a loss to explain the question of the Wassermann, unless it was due to a fault in technic. Sometimes the reaction would be negative and sometimes positive in the same case, and these errors occurred in the best hospitals. These reports varied so that they had caused one man in New York to refuse to accept a diagnosis unless he had made the Wassermann test himself,—and he was not a man with a misconception of his ability. He did not know whether the leucocytosis could be accounted for by malaria. Typhoid would account for it. He was sure that he had lost a number of his earlier cases because he had used too much drainage. For the last four or five years he had used a cigarette drain in acute cases, with the idea of getting, at the end of twenty-four or forty-eight hours, a fairly free channel running down to the abscess cavity. The cigarette drain could then be taken out and replaced by folded rubber tissue.

Dr. Ewing W. Day, Pittsburg, Pa., referring to the legal point raised in Dr. Richard's case, cited an experience along the same line. A patient was a minor and the father refused to give consent for operation. The boy was over sixteen and would not come under the judge of the juvenile courts. Three judges and the lawyer for the hospital all said he

would not be sustained in operating without the father's consent. He finally went to father's employer, who told him that unless he gave his consent he might look for another job.

Dr. James F. McKernon, New York City, cited a case in which the patient was of age and the father gave his consent, but the counsel for the hospital said if the patient lived and had any dissatisfaction he would have a "come back" against him and the hospital, according to the laws of the state of New York. The case described by Dr. Richards was one in which the result would have been the same no matter when the operation had been done, because an abscess, rupturing into the ventricle, was nearly always fatal. He called attention to a method of drainage with cargile membrane, which was allowed to remain until it was expelled by force of gravity or until the walls have collapsed back. There should be as little as possible of manipulation in abscess of the brain after the abscess cavity has once been evacuated. Otologists could learn much in this regard from the technic of the general surgeons, who simply incised, allowed the pus to run out, inserted a very small drain, and left it alone. The majority of their cases had done well in contrast to those of otologists in the past.

Dr. Wells P. Eagleton, Newark, N. J., said general surgeons have better results in brain abscess cases than do otologists because they do not have to deal with dirty mastoids, and if they did encounter such a mastoid they would leave it alone. Otologists would explore the mastoid, and then go on to explore the brain, and if they failed to find the abscess they simply condemned the patient to death. Dr. Richards had followed the proper course in his case. Operation early enough would have given the patient a good chance for recovery, because the abscess was of the kind mentioned by Ballance as having a stalk. Abscess of this type gave the best possibilities for recovery. Such abscesses were fastened down by adhesive meningitis, and every

manipulation which tended to break down the protecting adhesions was bad surgery. The general surgeon, before opening an appendix abscess, walled everything off with the idea of preventing general peritonitis. It was much more important that the otologist should wall off the pia-arachnoid. The peritoneum was in the habit, so to speak, of dealing with infection, but this was not true of the pia-arachnoid. The chronic abscess had a distinct wall of connective tissue which gave it an envelop; the acute abscess had no wall, although microscopically it had been shown that at an early stage a wall was beginning to form. If, in draining an acute abscess the pus was sucked out gently the cavity would gradually fall in, and after a short time there would be no cavity. A small drain introduced at this time would keep it so. It was very much better, in brain abscess cases, to do a great deal too little than to do the least bit too much; for this reason he would rather not drain at all than to put in a drain that acted as an irritant. Not only this, but in passing the probe there was danger of going into normal tissue and of generalizing a localized abscess.

Dr. Richards, in closing the discussion, agreed with Dr. Eagleton concerning the matter of drainage. The drains used were small collapsible tubes an inch or an inch and a half long. As the man died within thirty-six hours no harm could have resulted from any defect in drainage.

ACUTE HEMORRHAGIC MASTOIDITIS.

By HANAU W. LOEB, M. D., St. Louis, Mo.

It is rare indeed that an opportunity is afforded of discovering the presence of an acute hemorrhagic mastoiditis. This is only rendered possible by operating while its hemorrhagic nature is still manifest or by post-mortem examination where death has supervened from an intercurrent disease or from an early and fatal complication.

According to the usual rule, an infection resulting in hemorrhagic inflammation of the mastoid cells should be of a virulent nature and a serious result would be anticipated unless intervention is undertaken early in the disease.

The following case furnished an abundance of evidence in this direction: Miss Z. W., a pupil nurse at the Jewish Hospital, St. Louis, was taken on the evening of March 27th, 1914, with severe pain in the left ear. She had previously never had any ear trouble whatever and had never been subject to acute inflammatory troubles of the upper respiratory tract. She had had the usual diseases of childhood, however, without any aural complications.

On examination, the drum in the region of Shrapnell's membrane was slightly red, but not swollen or bulging. During the night, however, the membrana tympani ruptured and by morning, there was an abundance of serosanguineous discharge from the ear. The pain continued and pressure over the mastoid process was almost unbearable. Meantime, her temperature ranged during the day from 99° at 10 A. M., to 101° at 9 P. M.; pulse rate from 90 to 115. During the night the discharge was exceedingly copious, being still of the serosanguineous character and the pain continued severe.

On the following day, March 29th, the temperature and pulse range were about the same, lower in the morning and higher in the afternoon. The pain continued severe and the discharge became more copious, but maintained its serosan-

guineous character. Blood count 15,900, with 78 per cent. polynuclear neutrophiles, 11½ lymphocytes, 9½ monomuclears and transitionals, 1 polymorphonuclear eosinophiles. Smears from culture made from ear discharge showed Gram negative diplococci in short chains. Urine, specific gravity 1024, with a trace of albumen.

In view of the continuance of the pain and discharge, the sensitiveness of the mastoid process, the character of the organism, which by this time had been determined to be a diplostreptococcus of the type described by Davis except for its Gram negative stain, and numerous fatalities in throat infections, described by Davis, it was determined to exenterate the mastoid cells. This too in spite of the fact that less than four days had passed since the primary focus in the middle ear had manifested itself by pain.

After the customary preparation, a simple mastoid operation was made. The soft parts were not swollen and gave no evidence of any inflammatory or septic condition. When the periosteum was elevated, the bare bone appeared almost blue in color as if there had been marked necrosis present.

Upon chiselling away the cortex, this was found due to the presence of a reddish blue clot filling every cell that was opened. In fact it was possible to identify each cell by the color of the clot showing itself through the thin bone. No pus or granulation tissue was found in any of the cells, and the antrum was filled with a clot similar in character to that found in the other mastoid cells. The mastoid was entirely exenterated, the cavity in the bone packed with plain sterile gauze, and the skin incision closed by sutures, except at its lower portion which was left open for the gauze drain.

Temperature six hours after the operation 102.5. It continued close to this mark for eight hours when it began gradually to recede. It rose again to 101 during the second evening after the operation and to 101.5 on the following day, but after that, during the week following, it gradually approached the normal with slight elevation every evening.

On the day after the operation the blood count was 31,900.

three days later 23,000, a week after the operation 21,700. Blood culture which was made on the day of the operation proved to be negative. On the third day after the operation the urine was loaded with red cells, which however, soon disappeared. The healing was rapid except for a small sinus which persisted for several months.

The bacteriologic report made by Dr. F. L. Evans, is as follows:

"March 27th. Cultures show medium size opaque colonies, not mucoid and non-hemolytic. Smears from colonies show pure cultures of Gram negative diplococci, which have tendency to form short chains. Organisms are not encapsulated, but have a slight halo surrounding them. Smears from discharge show diplococci only, no chain formation.

March 30th. Mastoid operation. Culture from clots in superficial cells gave pure cultures of diplostreptococcus. Smears show numerous red cells, pus cells and diplococci. From inner blood clots of the deeper cells both diplostreptococci and staphylococci recovered. Hemolysis, capsulation and mucoid character not present.

April 1st. Diplostreptococci transferred to bouillon.

April 2nd. Bouillon growth almost exclusively diplococci, a few short chains however being found. 1/10 c. c. of bouillon culture is injected into ear vein of a rabbit three months old.

April 7th. Rabbit died during the day. Inflamed area around the site of injection, no sloughing. Culture made from heart's blood and site of injection. Intense hyperemia of all organs.

April 8th. From heart's blood. Gram negative diplococci with a slight tendency to form chains were recovered. Cultures from site of injection, negative.

April 9th. 1/10 c. c. of recovered diplococci (bouillon culture) injected into knee joint of rabbit.

April 15th. Area of inoculation shows necrosis, no pus; cultures contaminated. Animal sick.

April 18th. Rabbit died; cultures show staphylococci and hay bacilli."

It is fair, therefore, to assume that the acute hemorrhagic mastoiditis resulted from a middle ear infection due to the Gram negative diplococci in short chains such as were found in the original discharge from the middle ear, in the clots from the mastoid cells and the antrum, and which were fatal when cultures of the organisms were injected into a rabbit.

There were in the hospital at the time quite a few cases of diplostreptococcal infection in various manifestations, such as lacunar tonsillitis, acute endocarditis, acute articular rheumatism and purpura hemorrhagica, which will be reported in a later paper.

There are three interesting questions brought up by this case.

1. The microorganism.
2. The hemorrhagic character of the inflammation.
3. The early date of operation.

1. The organism differed from that described by Davis and Rosenow, which was (Journal of the American Medical Association, 1912. Vol. LVIII, p. 773) encapsulated and Gram positive, two factors which according to Rosenow may depend upon cultural conditions within and without the body. It differs materially from the streptococcus mucosus described by Neumann and Ruttin (Arch. fur Ohrenheilk. Vol. 79, 1909, p. 1,); in fact the clinical course of the case reported is entirely at variance with that of the type reported by these writers. Alberti (Arch. fur Ohrenheilk. Vol. 99, 1913, p. 111,) who made an extensive study of the bacteriology of acute mastoiditis, does not mention an organism possessing the characteristics of that which is here reported.

2. The hemorrhagic character of the inflammation according to the general rule would indicate a virulent type of infection. A fairly detailed study of the literature has failed to reveal instances in which other operators have

found a similar picture. The condition present could not have been anticipated by any of the symptoms unless the uncommonly long duration of the serosanguineous discharge might indicate a hemorrhagic exudate. It was exceedingly interesting to observe each cell come into view as the clot appeared visible through the thin bony wall of the cell.

It is quite possible that more of these cases of hemorrhagic mastoiditis would be found if operative interference were called for so early in the course of the disease.

3. The justification for operating so early in a mastoid affection may be doubted. However, an infective agent, unquestionably of high virulency, symptoms rapidly increasing in severity in spite of copious discharge, increasing leucocytosis and the compromising possibility of an acute meningitis, call for an operation in no uncertain tones. The post-operative fever and leucocytosis add weight to the evidence.

I have found it very difficult to get any definite information as to the frequency of early operations in mastoiditis following a first attack of acute suppurative otitis media. However, it is evidently exceedingly uncommon to operate as early as four days after the appearance of the initial symptoms. Several otologists have given instances to me of such early operations, but they have been, in the main, complications of scarlet fever, measles or grip.

Texier and Levesque (Rev. Hebdom, June 20th, 1914.) describe three types of mastoid disease: Subacute, acute and hyperacute. In the last, tympanic and mastoid cavity are invaded at the same time by a very virulent microbe which may kill the patient by infection, intoxication or meningitis before pus is formed in the mastoid cells. They have operated on some such cases. They claim operations should be performed two, three or four days after appearance of otitis, but they do not state how early they operated in any of their cases.

Taking the case as a whole, it demonstrates not only the occurrence of an acute hemorrhagic mastoid inflammation

of bacterial origin, but also that a mastoid infection may be coincident with as well as dependent upon a middle ear inflammation.

TRANSITIONAL EPITHELIUM IN THE RHINOPHARYNX.

By W. SOHIER BRYANT, A. M., M. D., F. A. C. S., New York, N. Y.

In an effort to determine the boundary lines between the squamous epithelium and the ciliated epithelium of the rhinopharynx, an examination was made of the rhinopharynges in the domestic rabbit (eight individuals), in the guinea pig (three individuals), in the domestic cat (four individuals), in the macacus and cebus monkeys (one each), and in man (twelve individuals; adults, children, infants and fetus).

The search for these boundary lines demonstrated the presence of a third variety of epithelium (mentioned by von Ebner) which, in the character of its cells, is intermediate between the squamous cells and the ciliated columnar cells. This epithelium which occupies the transitional zone between the epithelium of the oropharynx and that of the nasal fossae, is composed of cuboid cells with either imperfect cilia or no cilia at all.

In all the specimens examined, the squamous epithelium extends as far forward as the fossae of Rosenmuller, the intermediate zone of the epithelium occupies the region of the orifice of the Eustachian tubes, while the ciliated columnar epithelium extends a variable distance backward, approaching the Eustachian tubes.

The intermediate zone of the epithelium lies in a wavy ring around the rhinopharynx. It bends forward on the anterior and the posterior walls, and backward on the lateral walls at the attachment of the posterior faucial pillars. The zone includes interdigitations and islands of the neighboring varieties of epithelium.

The boundaries of the intermediate zone of epithelium vary in position, as stated by von Ebner, but these boundaries, I find, contrary to von Ebner, are constant, inasmuch as they vary between fixed limits. In the several species examined, the variations have specific limitations.

1. In the rabbit, the anterior border of the transition extends far in front of the Eustachian orifices. The posterior border reaches these orifices.

2. In the guinea pig, it ends anteriorly a short distance in front of the Eustachian orifices, and posteriorly a short distance behind these orifices.

3. In the cat, its anterior border crosses the Eustachian orifices, while its posterior border is a short distance behind these orifices.

4. In the cebus and macacus monkey and in man, the position of its anterior and posterior boundaries resembles very closely that found in the cat.

The intermediate zone is seen to extend farthest forward in the rabbit, and farthest back in man.

The figures presented here are diagrammatic, projectoscopic outlines of vertical antero-posterior sections of the head with the nasal septum preserved. The shaded lines represent the cut edges of the mucous membrane. The blue area represents the band of intermediate epithelium between the ciliated columnar epithelium and the squamous epithelium.

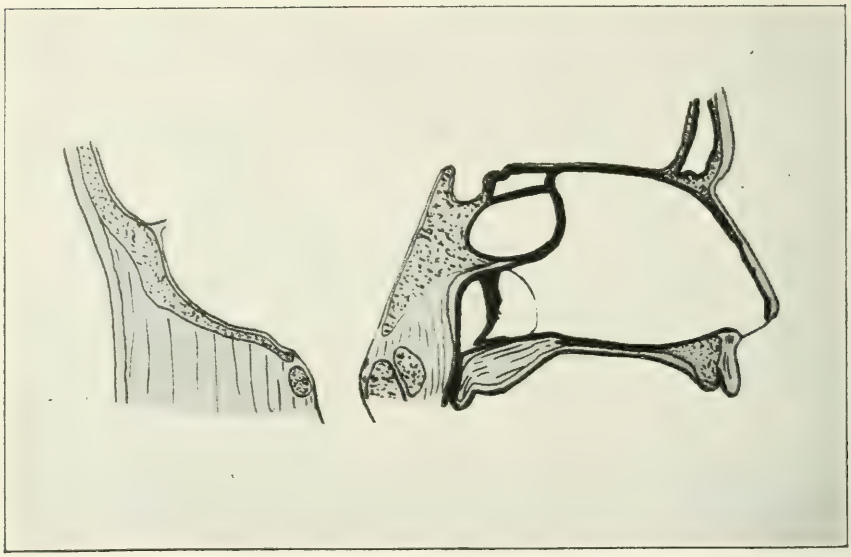


Fig. 1. Human pharynx. Adult.

The intermediate epithelial band (represented by blue area) extends forward on the back wall nearly to the septum, and backward nearly to the angle of the rhinopharynx. It passes over the Eustachian tube and inclines backwards at the union of the soft palate with the lateral wall. It then inclines forward to a point near the nasal septum on the floor of the rhinopharynx.

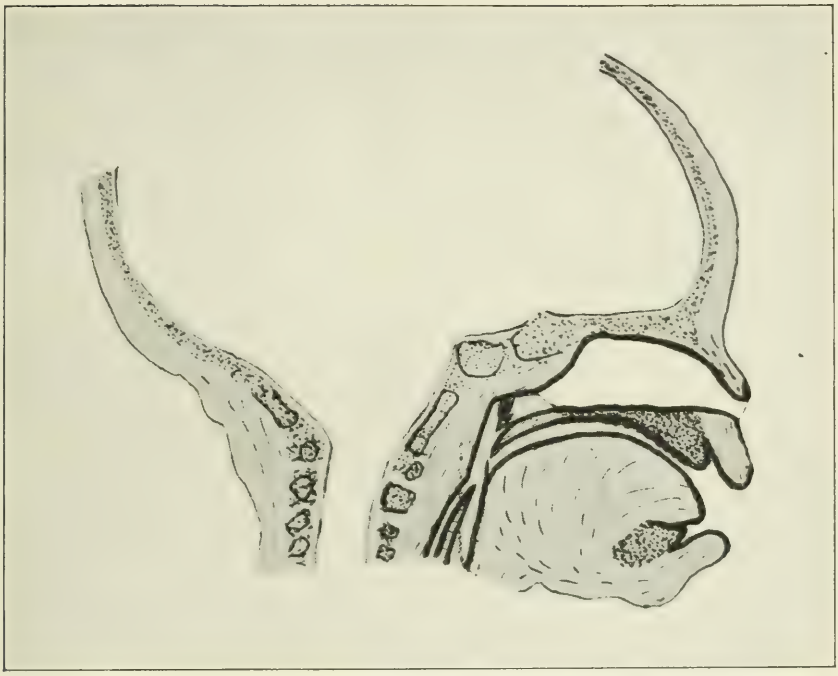


Fig 2. Human fetus at term.

The band of intermediate epithelium extends a little farther back than in the adult.

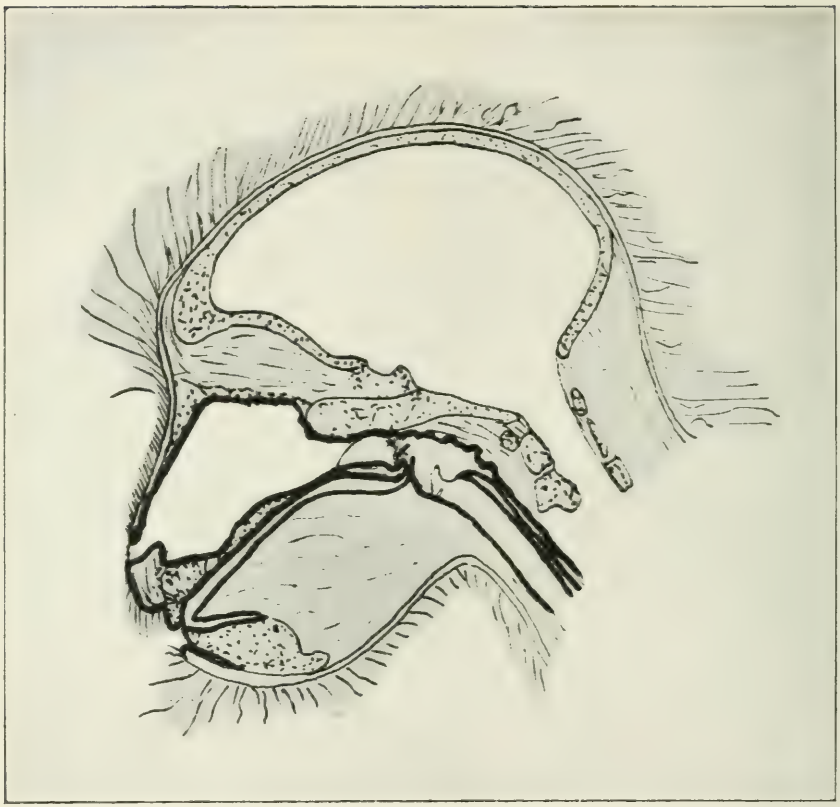


Fig. 3. Macacus monkey.

The band of intermediate epithelium lies behind the Eustachian orifice.

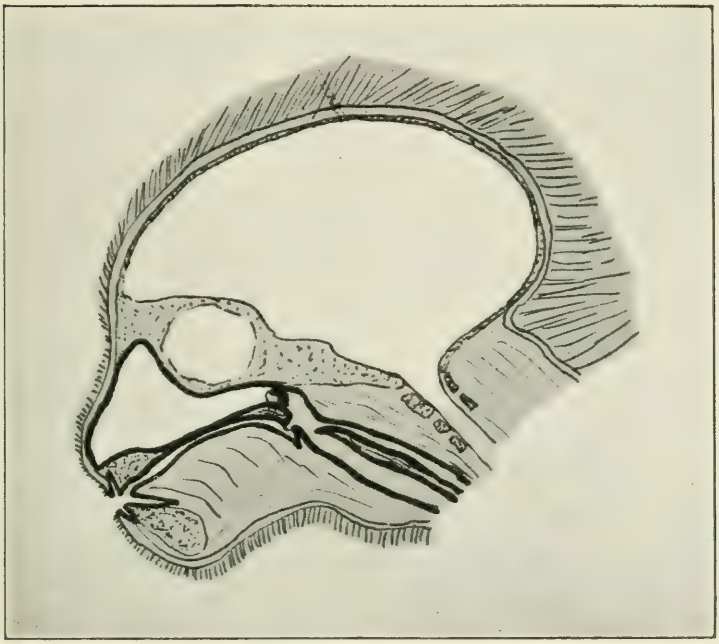


Fig. 4. Cebus monkey.

The band of intermediate epithelium lies behind the Eustachian orifice.

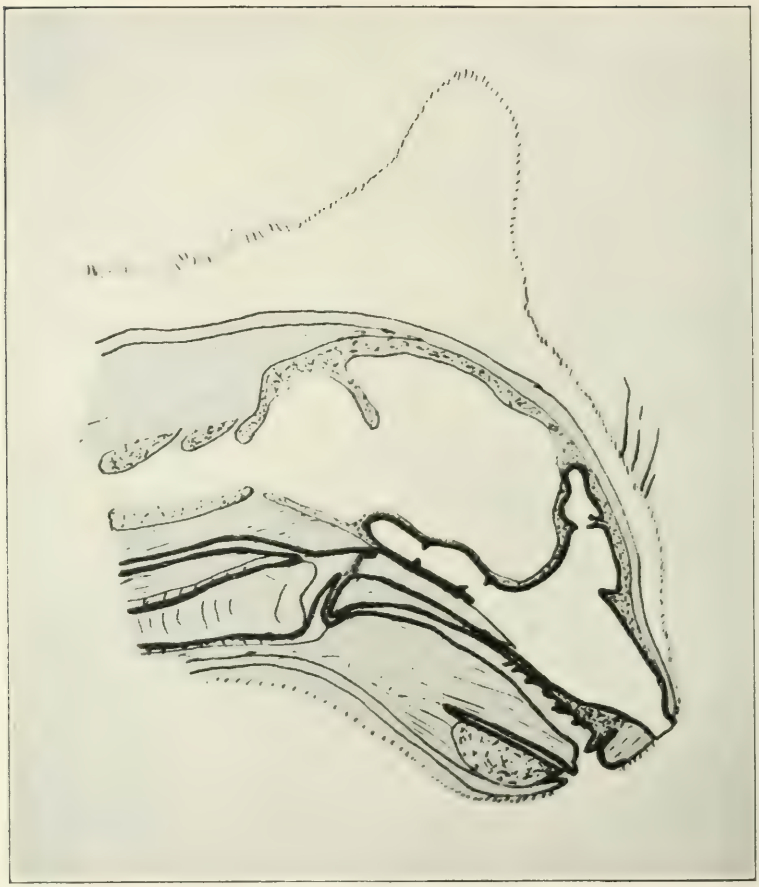


Fig. 5. Domestic cat.

The band of intermediate epithelium lies behind the Eustachian orifice.

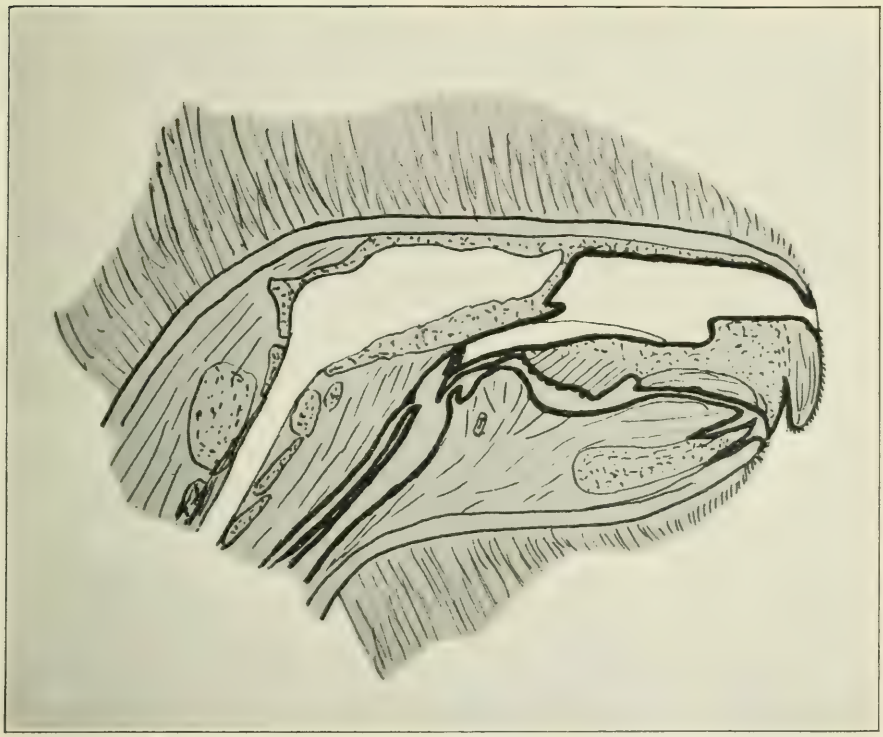


Fig. 6. Guinea pig.

The band of intermediate epithelium lies across the Eustachian orifice.

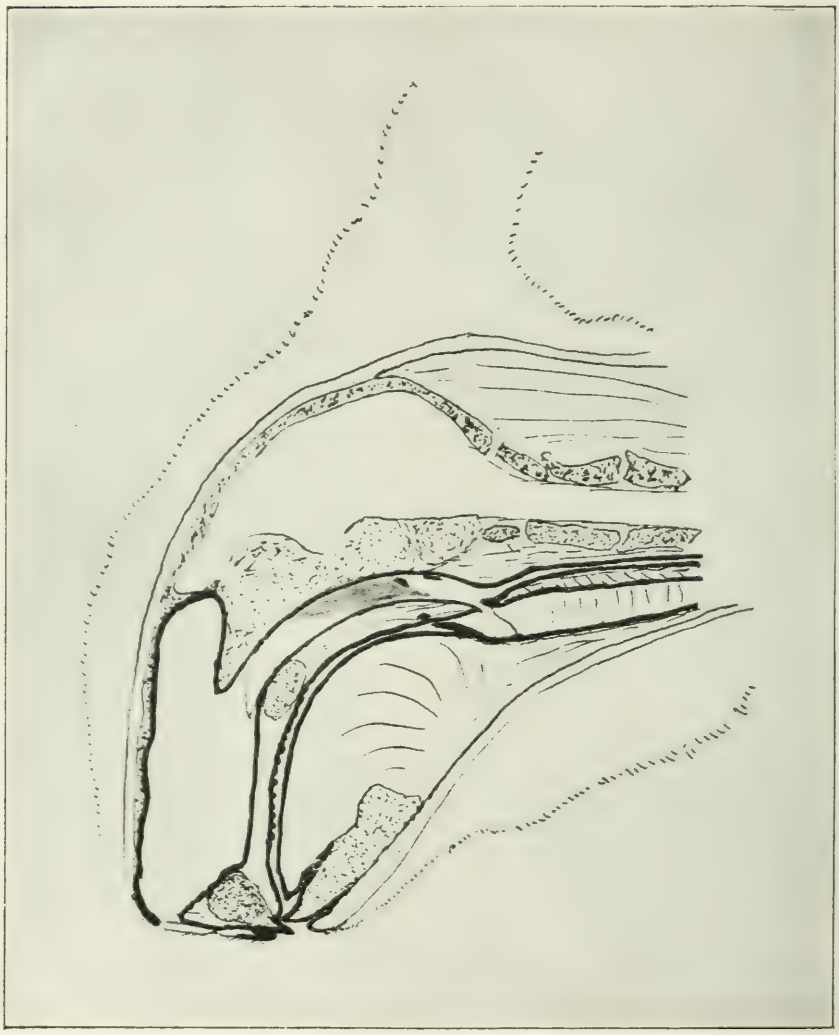


Fig. 7. Domestic rabbit.

The band of intermediate epithelium is wide and lies in front of the Eustachian orifice.

The following figures are microscopic sections stained with haematoxylon and eosine.

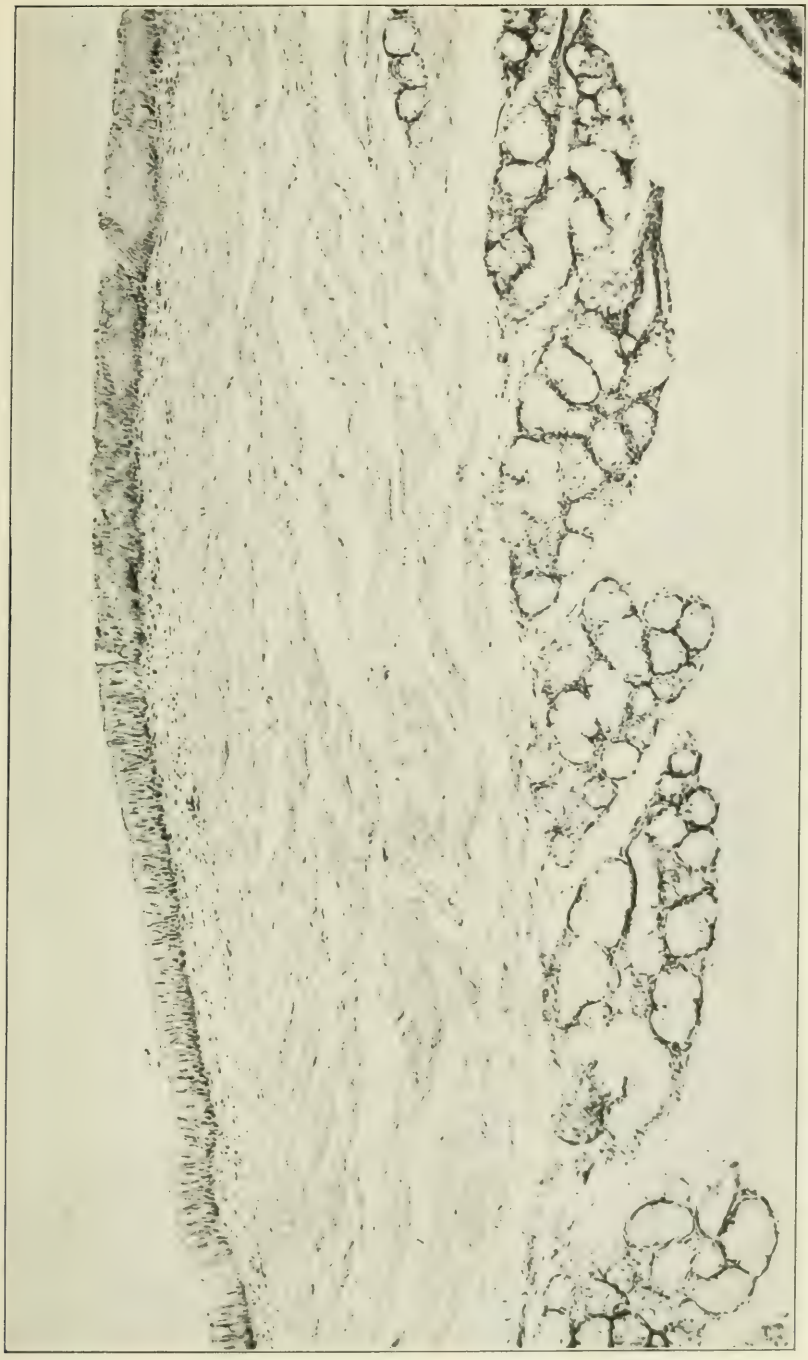


Fig. 8. Back wall of the rhinopharynx of rabbit shows transition of squamous epithelium on the right to ciliated columnar epithelium on the left. Mucous glands below.

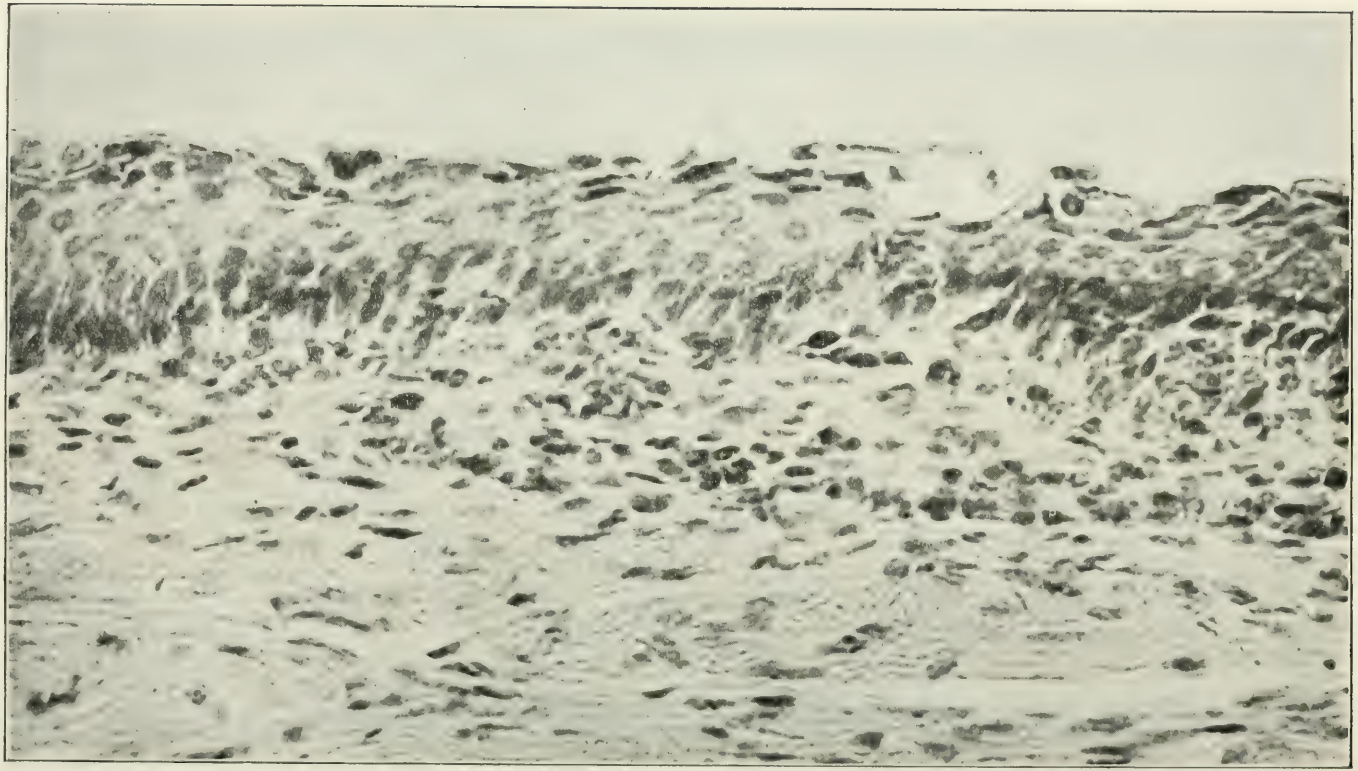


Fig. 9. An enlargement of No. 8, showing ciliated columnar cells on the left, changing to cuboid cells on the right. Rabbit.

Fig. 10. Epithelium from back walls of fossa of Rosenmuller, showing squamous cells on right and short columnar non-ciliated cells on left overlying lymphoid tissue. Transition of squamous to cuboid epithelial cells. Human.

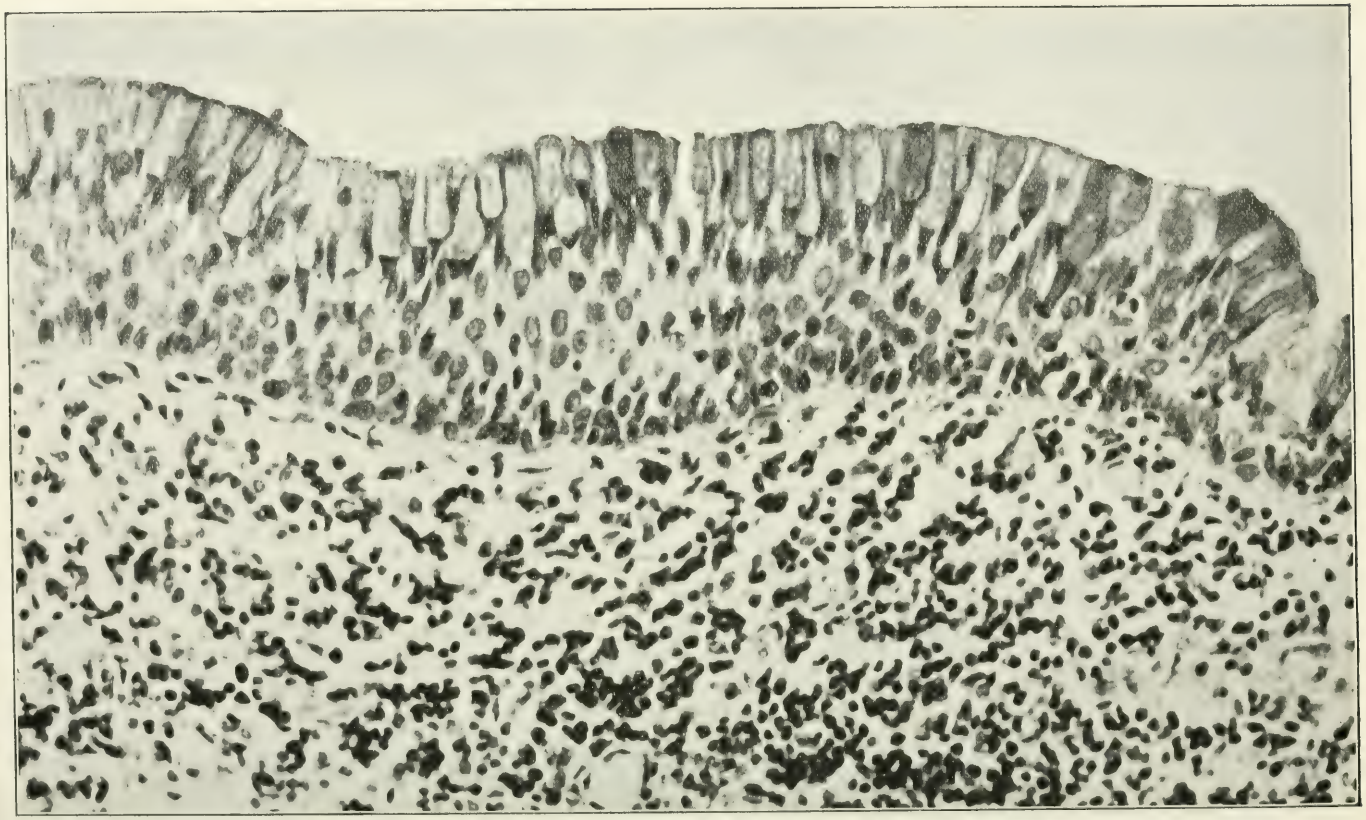


Fig. 11. Short columnar non-ciliated epithelium from back wall of fossa of Rosenmuller overlying lymphoid infiltration. Human.

The position of the transitional band obviously changes with the variation in the upper limit of the peristaltic digestive tube. We observe that it extends farther forward in the lower extreme of the mammalian scale, the rabbit, than in the higher extreme man; also that the width of the band is greatest in the rabbit, and least in man. Associated with these differences, possibly in a causative relation, the peristaltic digestive tube extends farthest forward in the rabbit, and farthest back, at its anterior end, in man. The posterior border of the intermediate zone lies nearly in the same position in all these forms of mammals, at a point where the mucous membrane begins to be exposed to food and digestive fluids. Its anterior border lies where the peristaltic motion starts.

This holds good for man, although in the adult we find there is a considerable area of squamous epithelium on the posterior pharyngeal wall between the posterior border of the transitional epithelial ring and the anterior commencement of the peristaltic tube. In the human fetus, this interval between the ciliated epithelium and the anterior end of the peristaltic tube, does not exist.

The position and extent of this transitional band is just what is required to cover the area between the free air portion and the repeatedly washed portion of the rhinopharynx; that is, the alternately washed and aired portion of the nasopharynx, between the portion which is only aired and the portion which is exposed to the materials of digestion. This area is, however, wiped at the time of swallowing, by the peristaltic contractions of the tube.

We readily appreciate the clinical and pathological importance which the unprotected area of nasopharyngeal mucous membrane presents in adult man. The area is covered by stratified squamous epithelium, but is not protected by the peristalsis of the tube.

Clinical experience has taught us that different kinds of epithelial surfaces do best with different kinds of treatment. The postnasal ciliated epithelium, the firm buccal stratified squamous epithelium, the thick soft esophogeal

stratified squamous epithelium etc., differ in resistance and reaction to re-agents. In the same way the nasopharyngeal ciliated epithelium, the intermediate epithelium and the rhinopharyngeal stratified squamous epithelium differ in sensibility and reaction. The ciliated aerial epithelium is very delicate and sensitive, reacting readily to very weak solutions, while the resistant thick stratified epithelium of the nasopharynx is as resistant almost as the mouth.

Furthermore, it has been observed, clinically and experimentally, that when ciliated epithelium has been destroyed over an area, it does not become wholly replaced, but its place is taken by stratified squamous epithelium which remains permanent.

These observations should warn us of the damage which is invariably done by strong re-agents to the ciliated region of the rhinopharynx.

The physiological protective mechanism of the mucous membrane of the rhinopharynx works in this way; the mucus, with adherent particles and fluids lying on the ciliated nasopharyngeal membrane, is wafted forward and expelled at the anterior nares. The fluids and solids and mucous secretions on the intermediate and squamous areas are squeezed backwards by the peristaltic action of swallowing.

This theory supposes that the peristaltic tube in the lower animals overlaps the ciliated tube. In adult man this overlapping does not exist, since there is a wide area of intermediate epithelium and squamous epithelium between the ciliated area and the commencement of the peristaltic tube. This area is dependent on the flow of mucus aided by gravity to cleanse itself. When the mucus becomes abnormally viscid retro-aspiration of the nasopharynx is resorted to. This action is useful in removing viscid mucus, but it roughens the cilia the wrong way to some extent. The pathological degenerate act of retro-aspiration of the nasopharynx exists in none of the mammals except in man.

It is obvious that this unprotected condition of the intermediate and squamous area anterior to the peristaltic tube in

man, offers a greatly diminished resistance to the entrance of all systemic infections, which usually commence at this point.

NOTES.

1. Von Ebner, Schlundkopf—Pharynx. Koelliker's Handbuch der Gewebelehre des Menschen, IX Edition, Leipsic, 1902, Vol. 3, p. 127.

2. These observations were made in the Pathological Laboratory of the Manhattan State Hospital through the courtesy of Dr. William Mabon, Superintendent, and Dr. Clarence O. Cheney, Pathologist.

SOME REMARKS ON THE X-RAY DIAGNOSIS OF MASTOIDITIS.*

By EMIL AMBERG, M. D., Detroit, Mich.

At the Ninth International Otological Congress at Boston, discussing Dr. Luc's paper, I mentioned the following case of otitis externa on the right side, in which the x-ray examination was of great help to me on account of the difficulty of the anamnesis.

No. I. Miss M. H., 15 years old, seen VI/18/12. Swelling over mastoid region on right side. No pain when auricle was drawn upward, forward and downward, but great tenderness on pressure over the whole mastoid process. The Roentgenologist reported the mastoid intact. I liberated, under gas anesthesia, about a teaspoonful of pus. Otitis Externa. The mastoid was not opened. Prompt recovery.

The assistance rendered me by the radiologist impressed me very much. I followed up the x-ray examination of the mastoid somewhat more systematically during the last winter. This work was facilitated by virtue of the fact that we have a very able Roentgenologist in our city. All the pictures were made by Dr. Hickey or his staff. As a rule both mastoids are examined.

No. II. Miss Fl. H., seen XII/5/14. Started with itching in right ear, external ear sore, thought to have mumps. Little serous discharge this a. m. Drummembrane appeared gray-red and bulging, tenderness over mastoid. Also otitis externa. Incised, pus. Yesterday felt dizzy and nauseated.

XII/6/14. X-ray examination. Cloudiness of right mastoid.

XII/7/15. From hospital record: The trouble began three years ago with pain in right ear and soreness of auricle.

Operation: Paracentesis. Diploetic mastoid, no free pus found.

Discharged XII/15/14, improved.

*Illustrated by lantern slides.

No. III. Mrs. T. Fr., 39 years old. Admitted XII/28/14. Suffered for several weeks from an otitis media suppurative left side requiring repeated incisions. Tenderness over mastoid.

X-ray: Compact left mastoid. Portion of cells cloudy.

Operation XII/30/14. Culture from mastoid. Short chained streptococcus. Free pus in tip.

Discharged I/14/15, improved.

No. IV. Girl H. Wh., 5 years old. Started I/20/15 with an otitis media and tonsilitis. Temperature 106°. Drummembrane incised the same day. Very free discharge, mostly streptococcus. Redness over mastoid I/23/14. On I/24/14 very distinct mastoid tenderness. X-ray picture same evening showed mastoid involvement. Operation same night. Free pus in large terminal cell. Pneumatic large mastoid. Temperature going down only gradually. Discharged II/5/15 improved. Prolonged convalescence, two large sequesters taken out of wound about nine weeks after operation. Recovery.

No. V. Boy D. K., 5 years. Admitted II/1/15. Otitis media supp. in left ear. Incisions I/27/15; I/28/15; I/29/15; last time in ether narcosis. Drummembrane sagging, thick. Severe headache. Some tenderness over antrum. X-ray picture: Mastoid cells clear except in region of antrum. Operation II/2/15. Pus in antrum.

Blood: Poly. 74%; Small 11%; Large 15%.

Culture from left ear.

Short chain streptococcus, non-encapsulated diplococcus which is diplococcus catarrhalis.

Discharged II/11/15, improved.

Remarks: The radiologist (Dr. Hickey was absent) reported the mastoid clear. I called his attention to the region of the antrum where a marked difference in favor of the other ear existed. Together with the clinical symptoms the condition appeared to require surgical interference. The finding during the operation proved the intervention to have been necessary.

No. VI. Mrs. G. B., 27 years old, seen II/7/15. Admitted II/7/15. Says ear began to ache II/4/15. Membrana tympani incised when seen II/7/15. Some edema over mastoid region; could open mouth only a little on account of pain.

X-ray examination II/8/15: Breaking down of cell walls. Mastoid region cloudy.

Blood examination:

Polymorphonuclears,	75%
Small lymphocytes,	10%
Large,	1%

Operation II/8/15. Large mastoid, thin cortex, pneumatic, half loose piece of bone on base of tip. Pus and granulation throughout mastoid. In region toward carotid artery, a gaping or tapping rhythmical noise heard (the only time the writer ever heard it).

Discharged March 2, 1915, improved.

No. VII. Mrs. M. D., 35 years old. Pain behind left ear about six weeks ago. Operation advised before, but patient was afraid and remained away from observation.

Admitted II/14/15.

X-ray examination: Lower left mastoid fused into one large cell. Reminder of cells cloudy.

Operation II/15/15. Upon incision through edematous tissue foul smelling pus; fistula at angle of mastoid process, and posterior wall of canal at about 2 o'clock direction. Large terminal cell; granulations, free pus through whole portion of mastoid as indicated in X-ray picture and about one-half inch inwardly.

Discharged II/24/15 improved.

No. VIII. Boy E. C., 7 years old, l. e., admitted II/19/15. From hospital record: Five years ago had scarlet fever. Since then, occasionally had discharge from ear. Sometimes, for periods, ear discharged continuously.

X-ray picture: The left mastoid looks affected.

Operation II/19/15. Bone inflamed, increased vascularity. Sclerosed toward antrum.

Discharged II/26/15, improved.

No. IX. Mr. J. P., 23 years old. From hospital record: Admitted III/5/15. Pain in left ear, Feb. 23rd, 1915; bloody discharge next day, discharge continues. Some tenderness over tip of mastoid since March 4. Patient had slight pain in chest before earache began. Polymorph, 79%; small 11%; large 10%.

X-ray examination: Right cells pneumatic throughout. Cells about the left antrum are sclerosed. Cells of left tip pneumatic.

III/7/15. Left drummembrane incised.

No mastoid operation.

Discharged III/15/15, improved.

Relapse, readmitted III/17/16.

Polymorphonuclears,	66%
Small lymphocytes,	25%
Large lymphocytes,	6%
Mastcells,	2%

Remarks: Patient has pronounced deviation of the septum to the left. Treatment besides douching, rest in bed. I follow, whenever possible, the practice of Heine-Munich, who recommends to keep patients with acute otitis media in bed. He claims that early incision of the drummembrane and rest in bed, even if the patients have no fever and object to this procedure, have given the best results. Heine adds (B. Heine, Operationen am Ohr, 1913, page 19) that no mastoid operation was necessary in private patients treated in this manner, although he thinks they may be necessary in rare instances (see our patient No. 5). These results, he quotes as proof, are different from those in the polyclinic. I may add, the general practitioner or surgeon keeps patients with appendicitis in bed.

No. X. Dr. A's son, 4 years old. Otitis media acuta, incised III/9/15, and III/12/15. Apparently tenderness over left mastoid.

X-ray: Mastoid clear; no operation; recovery.

No. XI. Mr. N. F. From hospital record: Admitted III/29/15. Present illness began ten days ago in a severe

cold. Patient noticed pain in each ear. Five days ago pain in ears became more severe and discharge started from each ear. The discharge has been profuse. Respiratory: Depression in infra and supra clavicular spaces. Mucous rales over anterior aspect of chest.

Blood count:

Poly,	79%
Small,	12%
Large,	9%

Patient had considerable headache, and some tenderness over both mastoids.

X-ray III/31/15. (See 11 a).

Examination of the right and left mastoid cells shows symmetrical condition of the cells of the two sides. On the left side the cells extend much further forward, some of them being anterior to the auditory canal. We note that in the upper cells in the posterior part of the left mastoid there seems to be a diminution in the sharpness of outline, which would be suggestive of a very mild infiltration. The septa, however, can be distinctly made out. We would think there was no destructive process.

On the right side we note that there is a very large indistinctness which is not so pronounced as on the left side.

Urine: Albumen III/30/15. No albumen IV/2/15.

Discharge from ear, culture: Staph. aureus, diplococcus catarrhalis.

Temperature between 100° and 98°, of septic character for a few days. IV/3/15 left M. T. incised.

Discharged IV/7/15, improved.

Readmitted to Harper Hospital IV/19/15, now complaining of pain and tenderness in right mastoid region. Tenderness about one inch behind right auricle.

Operation IV/19/15. Mastoid diploetic. Pus in mastoid. Sclerosed toward antrum. Prompt improvement. Discharged IV/27/15.

On V/1/15 pain developed in left mastoid in a spot about three-quarters of an inch behind left auricle.

5/1/15 x-ray picture shows mastoid involvement. (See 11b.)

Operation 5/3/15. Bone toward antrum sclerosed. Abundant pus in body of mastoid, far back. Recovery.

Remarks: The two pictures of the left mastoid allow a comparison concerning the progress of the affection. This shows the value of repeated x-ray examinations. The ideal way would be to have an x-ray record of the mastoid during health.

No. XII. Mr. J. D., 42 years old. Admitted III/8/15. Complained of pain on right side of head, apparently emanating from an infiltrated spot below and behind the right mastoid.

From hospital record: History of gonorrhea and syphilis about twenty years ago.

Present history: Began one month ago with cold in head followed by earache for about two weeks, then pain began behind ear and upwards. Had severe headaches for a long time. Blood examination:

	3/8/15	3/24/15
Polymorphonuclears.	80%	89%
Large lymphocytes.	8%	3%
Small lymphocytes.	12%	8%

X-ray 3/9/15. Left mastoid compact with exception of small mass of cells near antrum. Right mastoid has the same formation but the cells near the antrum have broken down, forming a comparatively large cavity. The region is cloudy, suggesting infiltration of this portion of the mastoid.

First operation III/9/15. Tip of mastoid softened. Infiltration below mastoid persisted.

At a second operation III/12/15, the middle cranial fossa was exposed and the antrum opened very widely. Headache persisted. A neck abscess was opened III/24/15 under local anesthesia by Dr. Ballin. Temperature during whole stay in hospital between 101° and normal. Wassermann (blood) reaction negative. Clinically syphilis, report of

dermatologist Dr. Varney. Antisyphilitic treatment makes headache disappear. Recovery prompt.

No. XIII. Dr. M.'s son, 5 years old, seen IV/11/15. Left ear had been incised. Incised again April 15th, twice. Mastoid tenderness developing.

X-ray. First degree mastoid; no operation; recovery.

No. XIV. Mrs. J. B., aged 56. Admitted IV/20/15. Pain and tenderness in left mastoid about six weeks. Discharge in left ear over four weeks. Swelling above and behind left ear. Urine acid 1038; albumen faint; sugar present.

X-ray picture shows mastoid involvement.

Operation IV/21/15, under gas anesthesia and locally ½ of 1% novocain subcutaneously. Mastoid cells appeared affected. Two counter-openings in edematous tissue above mastoid.

Discharged V/1/15, improved.

From the literature the following may be mentioned: Politzer remarks (Textbook 1908) that x-ray examinations are indicated in the following pathological processes:

(1) Foreign bodies (projectiles). (2) Injuries (fractures of the base). (3) Malformations. (4) Hyperostosis. (5) Destructions: By inflammatory processes, cholesteatoma, sequesters or tumors. In the latter it is especially important to see whether they reach the dura of the middle or posterior cranial fossa (resp. the sinus). H. E. Kanasugi differentiated with the aid of roentgenoscopy the pneumatic from the diploetic mastoid. He confirmed Politzer's findings that small and short mastoids show a diploetic structure and large mastoids a pneumatic structure and that a favorable relation concerning the location of the lateral sinus exists in pneumatic mastoids.

Rudolf Leidler (Clinical Roentgen Findings in Ear Patients) reports the findings in four patients in the Archiv. fuer Ohrenheilkunde, Vol. 85, I and II parts. In a male 60 years old there was on the right side incomplete peripheric facial paralysis and severe pain in the ear radiating toward the neck and head, especially severe when swallowing; no

mastoid tenderness on pressure, but granulations in the tympanic cavity. Histologically they consisted of flat epithelial cells. A diagnosis not possible (Prof. Albrecht). Only the x-ray picture allowed the diagnosis and topographical location of the tumor.

Case II. Female, 68 years old, suffered from a carcinoma emanating from the tympanic cavity. This was diagnosed also clinically, yet the size and topography of the tumor was only recognized by the x-ray.

Arthur H. Cheatle, London, at the Ninth International Otological Congress, reported an examination of both temporal bones from 120 individuals in reference to the question of symmetry in health and disease, without reference to x-ray examinations. This report is very important because we assume that, as a rule, the mastoid processes are similarly constructed on both side. Speaking under the heading of A., In Health; 2, The Interior, Cheatle says: "Classification of the types is founded on the condition of the outer antral wall and mastoid process. In referring to the mastoid process the terms 'upper and lower mastoid' are used, the 'upper mastoid' being the bone below the level of the apex of the antrum down to the upper level of the projecting part or 'lower mastoid.' On this basis it is found that of the 120 sets, 82 are symmetrical and 38 asymmetrical. In many of the asymmetrical bones there is a tendency to symmetry. It may be here stated that there are very few cellular bones without remains of diploe, either as a small mass at the extreme tip of the mastoid process, or as a rim around the tip." As examples may be mentioned asymmetrical, e. g.:

(b) Diploetic infantile type on one side. Dense outer antral wall with a few cells in the upper part of a diploetic mastoid on the other.

(c) Diploetic infantile type, on one side; dense outer antral wall, cellular upper mastoid and diploetic lower mastoid on the other.

(d) Diploetic infantile type, on one side; cellular outer antral wall and upper mastoid and diploetic lower mastoid, on the other.

(e) Diploetic infantile type on one side; cellular outer antral wall and entire mastoid on the other.

C, d and e explain why, in acute bilateral antral infection a mastoid abscess may result on one side and a chronic discharge without a mastoid abscess on the other. Those specimens which have only a dense outer wall and very few cells in the upper part of a diploetic mastoid have the same surgical importance with regard to the suppuration as the pure diploetic type, and the same applies to those bones in which the entire mastoid is composed of cells surrounded by a thick layer of dense bone. There are, therefore, twenty sets in which the diploetic infantile type is seen on one side only.

Later on, Cheatle states, under the heading "In Disease," that it can be pointed out to those who hold that the dense outer antral wall is the result of chronic suppuration, that the condition is present in this series without any signs of suppuration, on both sides, in 18, and on one side in 16. He adds that it can be confidently expected that in future x-ray photography will be of the greatest value in the diagnosis and treatment of suppurative disease.

Leidler and Schueller (The Anatomy of the Human Temporal Bone in the Roentgen Picture.—Archiv fuer Ohrenheilkunde, Volume 82, parts 3 and 4), comes to the following conclusions:

"The mastoid process is always distinctly recognizable. We can see its shape, whether long or short, broad or narrow—see whether it is diploetic, mixed or pneumatic. In the two latter instances we can form a judgment concerning the size and arrangement of the cells and their extension in the rest of the temporal bone (squama and pyramid). We further can see whether the corticalis is thick or thin, and in many cases we can announce the thickness in millimetres. A terminal cell, if present, is mostly plainly visible. The Roentgen picture gives us a definite idea concerning the shape, size and position of the outer auditory meatus. It mostly appears as a round or oval lightened area (Aufhellung) of a variable size, in which one sees the details of

the inner auditory meatus and sometimes also of cochlea. On three sides, namely, anteriorly, inferiorly and posteriorly, it is surrounded by the os tympanicum, which is always plainly visible and has a typical shape in the adult which is characterized by a part adapting itself bow-like around the ear canal and a continuation which goes parallel to the mastoid process and is of various width and length. Only in Specimen 6, the anterior portion of the os tympanicum passes the auditory canal. In children this bone forms a more or less broad ring.

The petrous bone represents itself as a triangle, sometimes long and narrow, sometimes short and broad. One can plainly recognize whether it is constricted or dense, or spongeous bone, and how large the labyrinthine kernel is. In children the pyramid forms an oblong, narrow triangle. If we now turn our attention to the finer formation of the labyrinth we must see that one can recognize always the inner auditory canal. The vestibulum in twelve, the semicircular canals in seven, and the cochlea in one hundred and thirty-three specimens. *The topographical relation of the inner auditory canal, the vestibulum and the cochlea is, in children and in adults, constant. The vestibulum always borders posteriorly and upwardly upon the inner auditory canal, whereas the cochlea surrounds it anteriorly and inferiorly.* The inner auditory canal forms a lighter field (Aufhellung) which is mostly sharply defined in the posterior upper part of the auditory canal, which frequently extends beyond the contour of the same. The size of the same varies. It reaches, however, at the most, one-third the size of the outer ear canal. Once in a while the inner auditory canal contains a picture which cannot be explained at the present time. In Specimen number one the inner auditory canal is exceptionally more visible in the anterior upper border of the outer external canal. Nevertheless, in this case also the topographical relations between the inner auditory canal, the vestibulum and cochlea are the same. The vestibulum in unfilled specimens shows itself as mostly little differentiated, more or less sharply outlined light field (Aufhellung) upwards and

behind the inner auditory canal, which never reaches the clearness of the latter. In size and shape varying, it is sometimes in children very large, sometimes only recognizable as a small strip. Frequently its size is determined by the attachments of the semi-circular canals. A distinct influence of the quality of the bone (whether diploetic or pneumatic) concerning the visibility of the vestibulum, cannot be shown.

The semicircular canals, which are plainly visible in temporal bones of children, can be seen in adults only imperfectly. Mostly the posterior semicircular canal is seen. The possibility to see the semicircular canal seems to be greater in pneumatic than in diploetic temporal bones. Of our specimens, five pneumatics show three, four diploetics only one semicircular canal.

The cochlea in unfilled specimen can always be seen in children, rarely in adults. It forms a lightened field (see specimen 1-7-11) of varied size anteriorly and below the inner auditory canal, lying within the contour of the external auditory canal, and usually of weak depiction. It cannot be shown that the quality of the bone has any influence upon its visibility. Five pneumatic temporal bones show the cochlea twice,—four diploetic the cochlea once.

The region of the tegmen, i. e., where the contour of the upper surface of the pyramid meets the squama, shows pretty variable conditions. Occasionally the thickness and position of the tegmen can be determined directly. (Frequently in children.) Mostly, however, one sees two contours, of which one corresponds to the upper edge of the pyramid, the other to the projection of the upper surface of the pyramid (frequently as a hunch superimposed upon the latter). The antrum shows the most variable findings in the Roentgen picture. It is mostly plainly visible in young children, forming a well outlined, relatively large lighter area within the pyramid backward and upward of the outer external canal, reaching frequently to the tegmen. In adults the findings vary from perfectly plain visibility to entire invisibility. *The quality of the bone is of essential influence concerning the visibility. In five pneumatic temporal bones unfilled, one*

only, in four diploetic ones four show the antrum. The antrum shows mostly a more or less plain lighter field within the pyramid. Mostly it is not sharply outlined and shows a weak depiction. The position of the lighter field and of the antrum in general is also not constant. Among nine filled specimens the antrum is found five times anteriorly and upwardly of the labyrinthine kernel in the pyramid and reached to the tegmen. In one case it takes the whole width of the pyramid above the labyrinthine kernel, and reaches from the contour of the posterior to that of the middle cranial fossa. In three cases it lies within the spongiosa. *All in all, we must say that the antrum that is not directly visible must be looked for mostly anteriorly and above the labyrinthine kernel near the region of the tegmen.* In two specimens (unfilled) also the attic is visible as a distinct narrow lighter field between the upper contour of the outer external canal and the tegmen. In four filled specimens it is visible always in the same location, always in connection with the antrum.

The sinus is plainly visible in all specimens. It possesses various widths and various degrees of curvature. *At present one cannot form a certain judgment concerning its depth, but the degree of the intensity of the lighter field is proportionate to its depth, so that one can draw the conclusion that a very intense lighter field speaks for a very deep sinus. Also the* appearance of the jugular bulb frequently allows us to draw an inference concerning the depth respectively of the forward position of the sinus. The bulbus is frequently visible as a more or less plain lighter field in the spongiosa of the pyramid in the posterior angle of its triangle. Not infrequently it also shows an arched outline. One can draw an inference concerning the height of the bulbus from the height of this lightened field, respectively of the curvature of the arch in the pyramid, and also concerning the depth of the sinus, because a high bulbus corresponds also to a deep sinus. Among eleven temporal bones of adults the bulbus could be plainly seen eight times. Sometimes the bulbus approaches the lower border of the outer external canal. In specimen number one the contour meets the posterior lower

quarter of the outer external canal. It cannot be shown that there is a difference in this respect in pneumatic or diploetic bones. The emissarium mastoideum is always plainly visible.

Ingersoll, in a paper entitled "The Value of Stereoscopic Radiographs of the Head," read before the Academy of Ophthalmology and Oto-Laryngology, 1914, claims that stereoscopic radiographs of the mastoid give us very much positive information in regard to pathological conditions. At the same meeting Dr. J. J. Kyle, of Los Angeles, says:

"As a confirmatory evidence of the existence of an acute mastoiditis, the value of radiography cannot be over-estimated. The findings are as positive as in nasal sinus disease and even more so. In a case in which the operator is inclined to procrastinate or feel over-conservative or in doubt, a radiograph should be made."

Conclusions.

1. The value of the interpretation of x-ray pictures of the mastoid affections would be materially enhanced if we had the pictures during health for comparison.

2. Repeated x-ray examinations are of great assistance.

3. The findings especially of Cheatle are an important factor in the interpretation of x-ray pictures.

4. In many cases the x-ray picture is undoubtedly of great help in the diagnosis.

5. An x-ray picture gives us an idea of the extent of the mastoid cells and of the character of the mastoid.

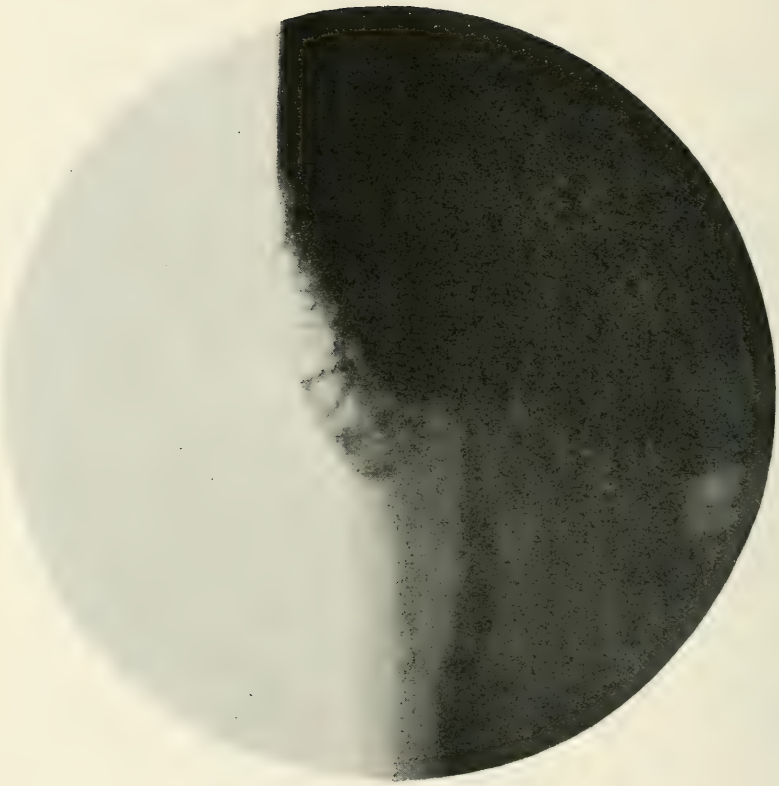
6. Some changes in the picture may be expected in any middle ear suppuration.

7. It seems not possible to differentiate sufficiently the changes which may be found in any middle ear suppuration from those indicating more serious involvement. Therefore, the clinical symptoms are of paramount importance.

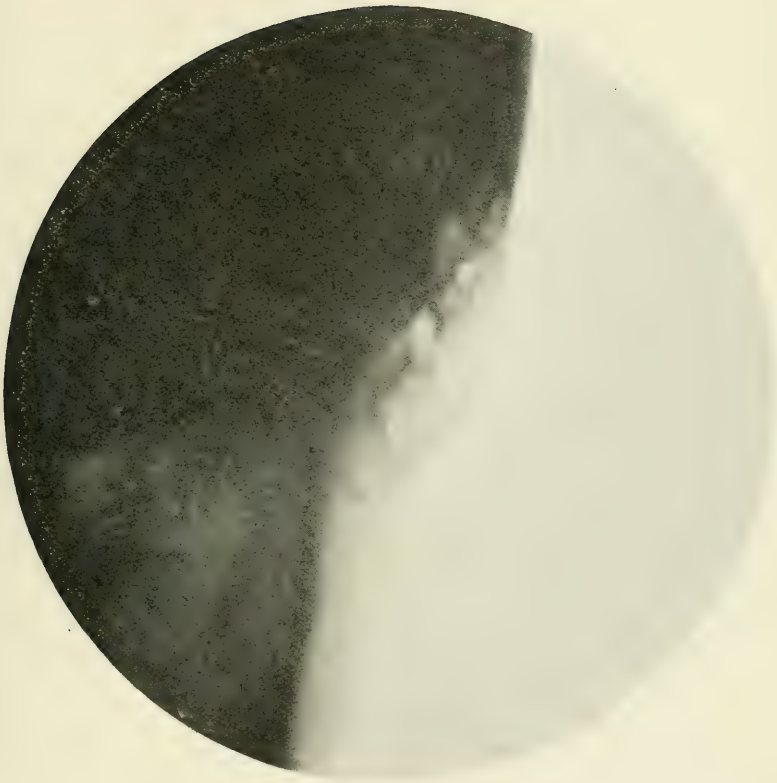
8. A negative x-ray picture of a patient with pronounced clinical symptoms must not be relied upon as excluding the seriousness of the affection and its complications.

9. The x-ray examination should be added to the routine examination of the mastoid.

The numbers underneath the plates refer to the cases of the author, not to the cases mentioned in the bibliography.



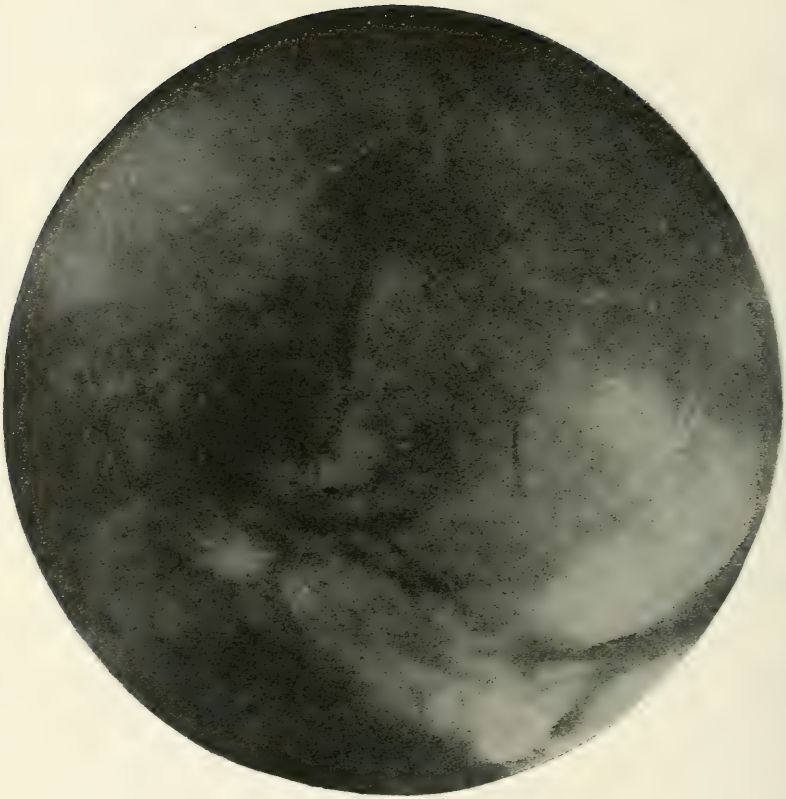
No. 1 (Left).



No. 1 (Right).

No. 2 (Left).

No. 2 (Right).



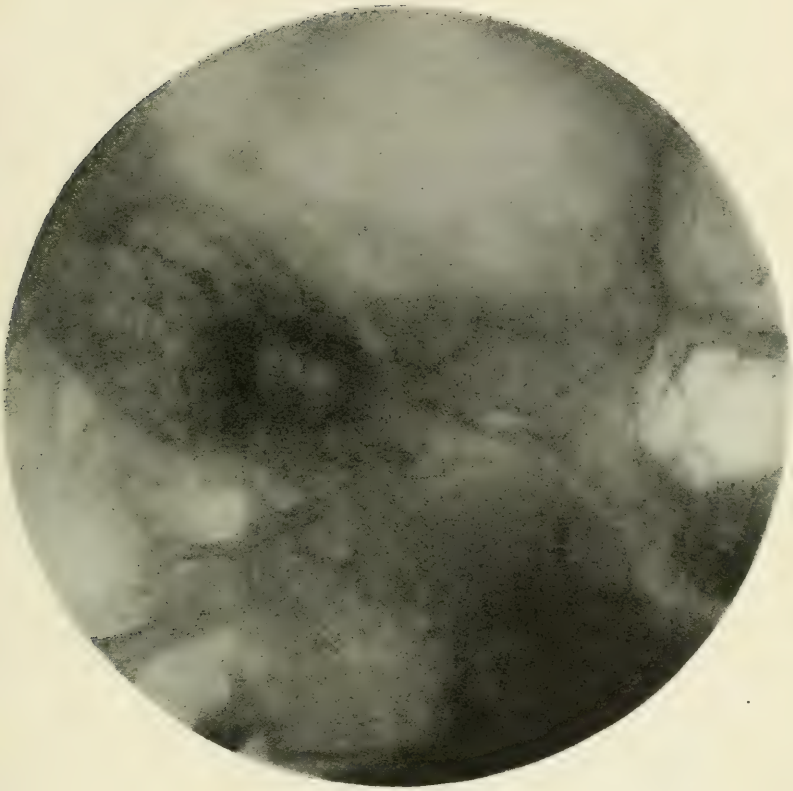
No. 3 (Left).

No. 3 (Right).

No. 4 (Left).

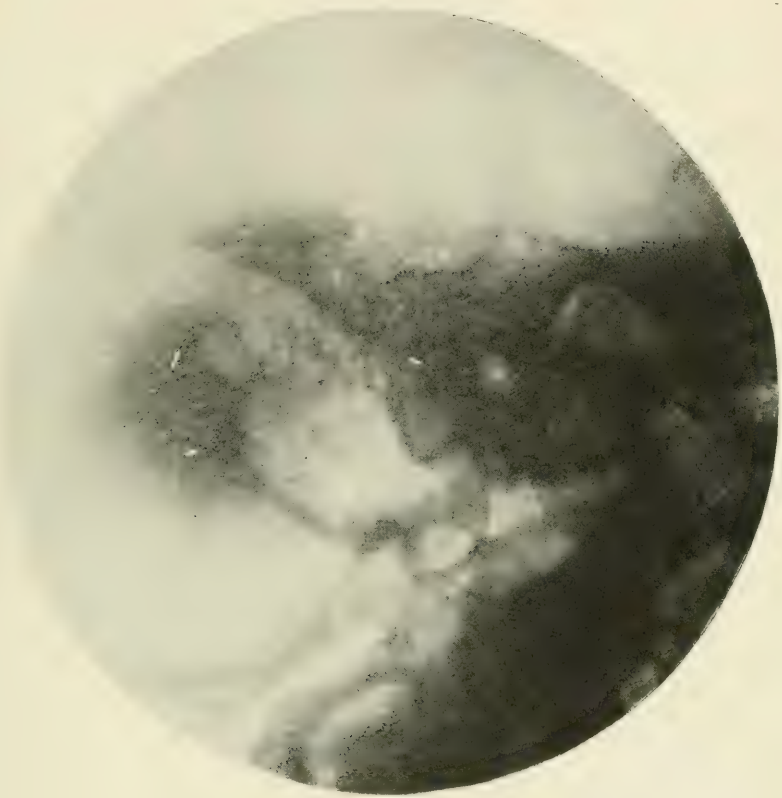
No. 4 (Right).

No. 5 (Left).



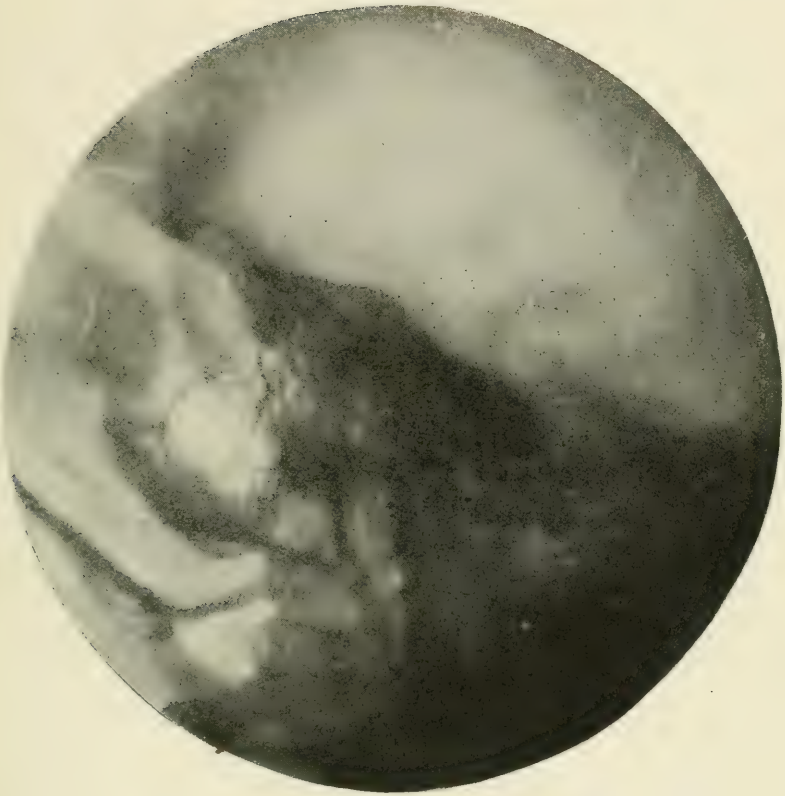
No. 5 (Right).

No. 6 (Left).



No. 6 (Right).

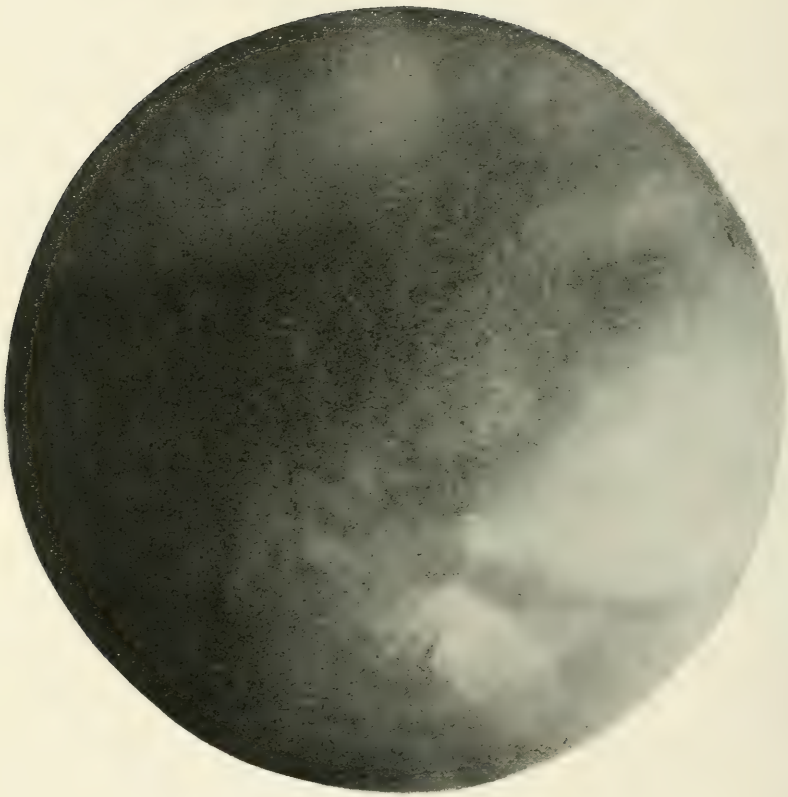
No. 7 (Left).



No. 7 (Right).

No. 8 (Left).

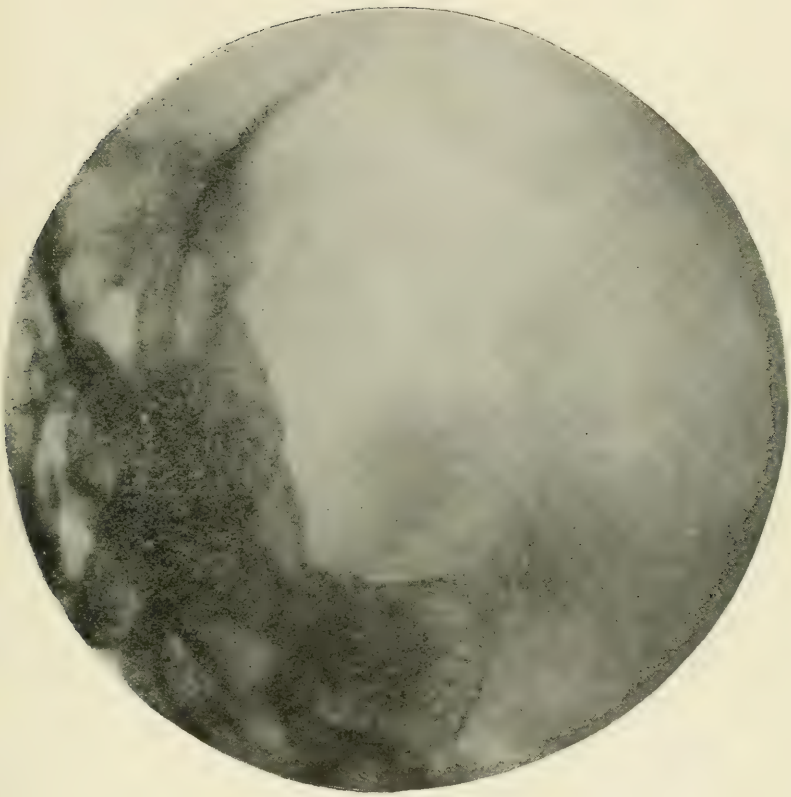
No. 8 (Right).



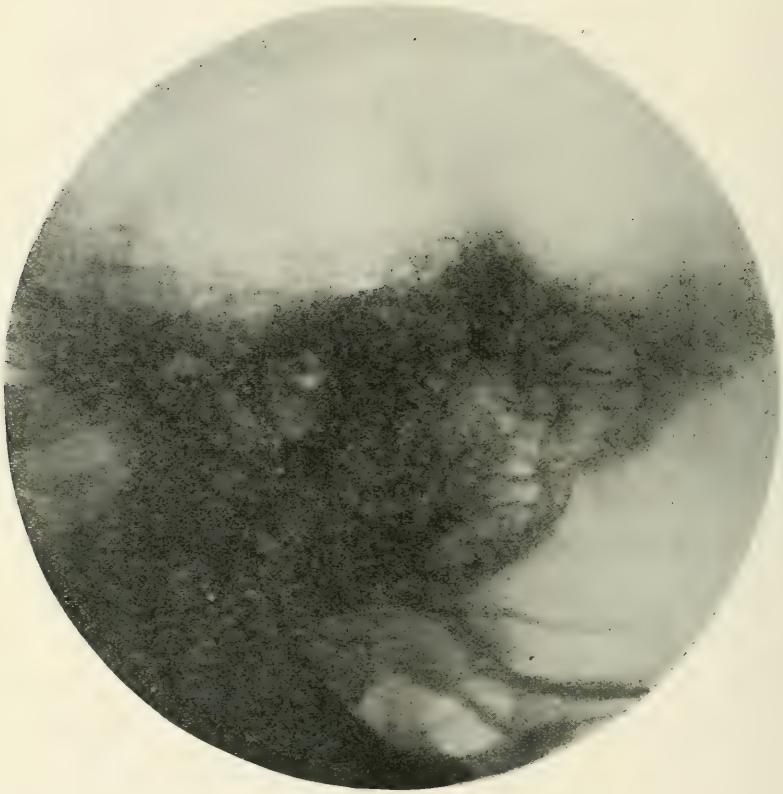
No. 9 (Left).

No. 9 (Right).

No. 10 (Left).

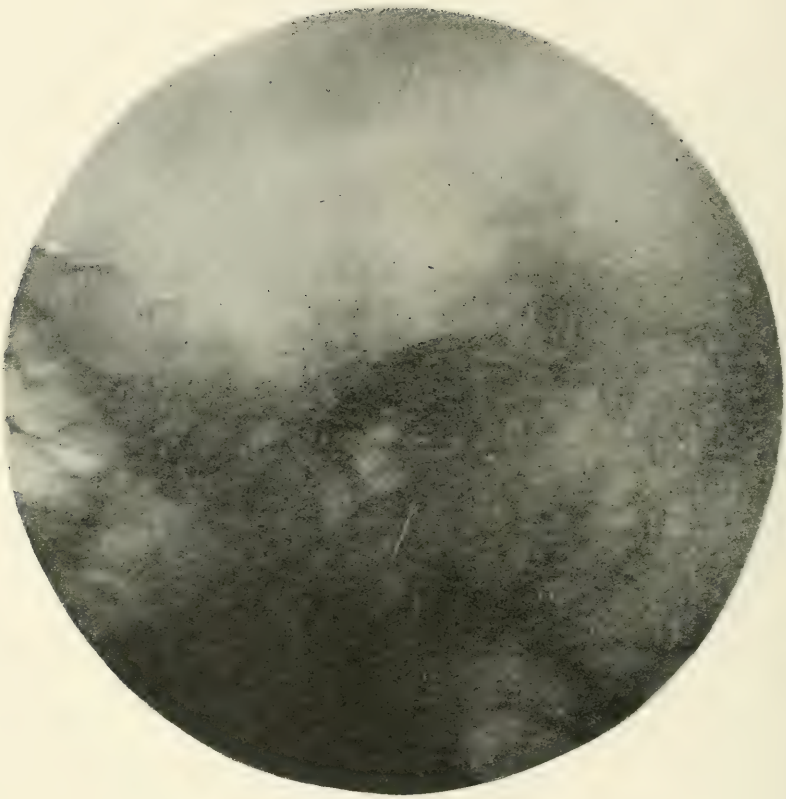


No. 10 (Right).

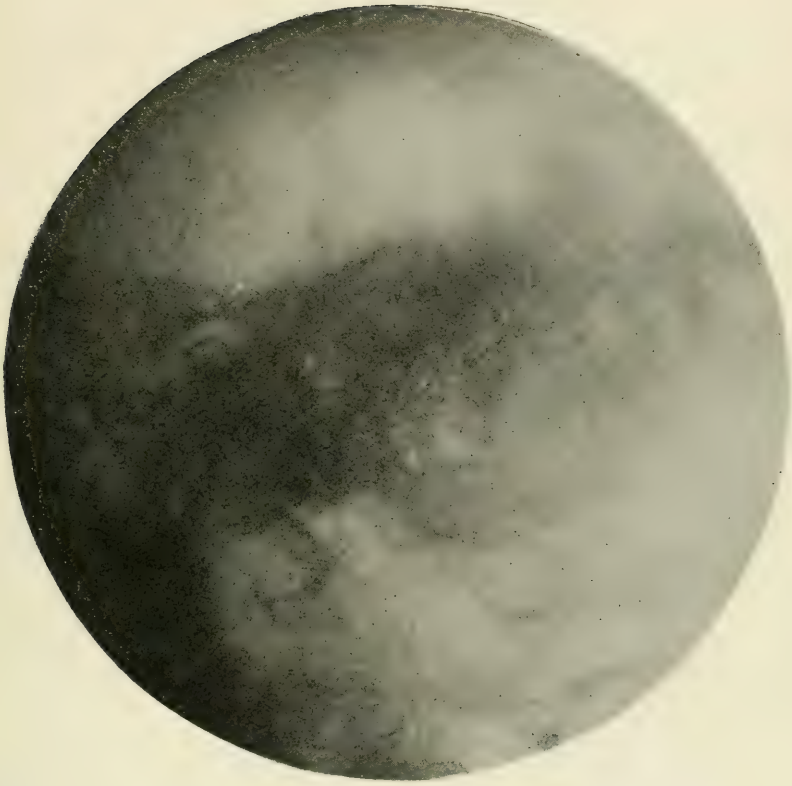


No. 11a (Left).

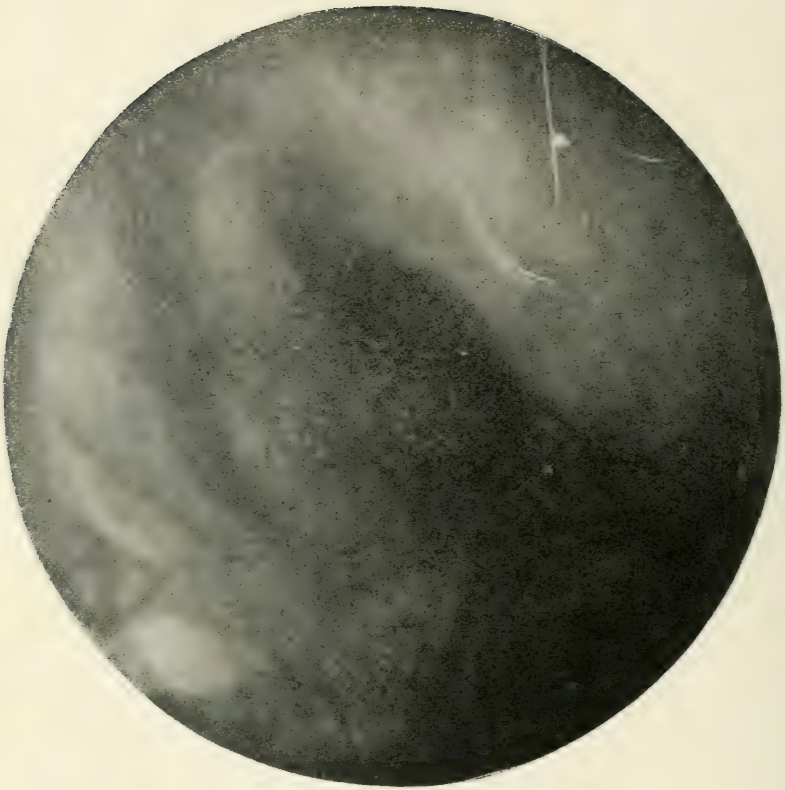
No. 11a (Right).



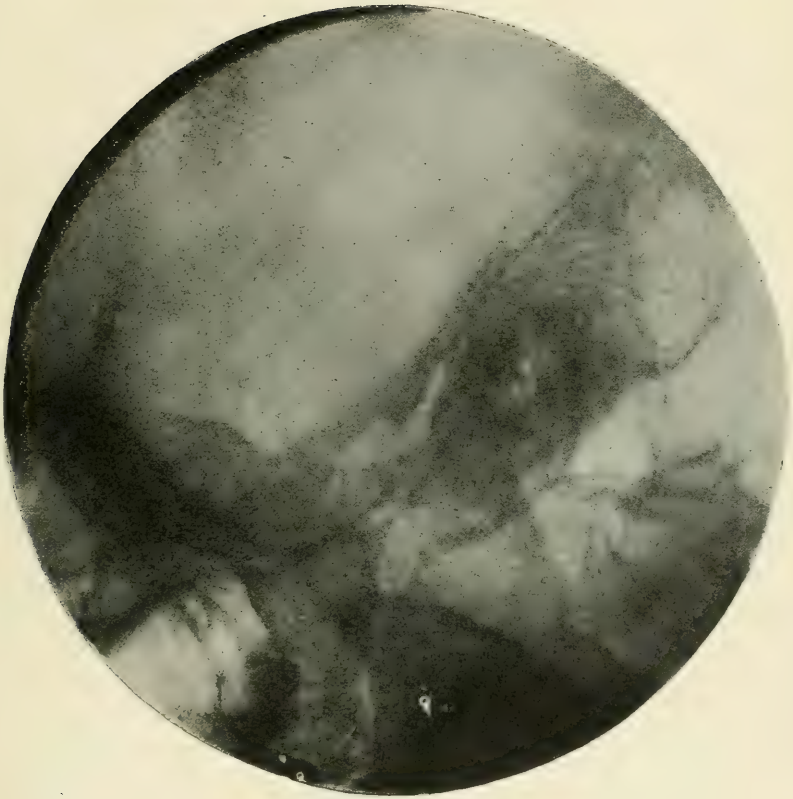
No. 11b (Left).



No. 12 (Left).

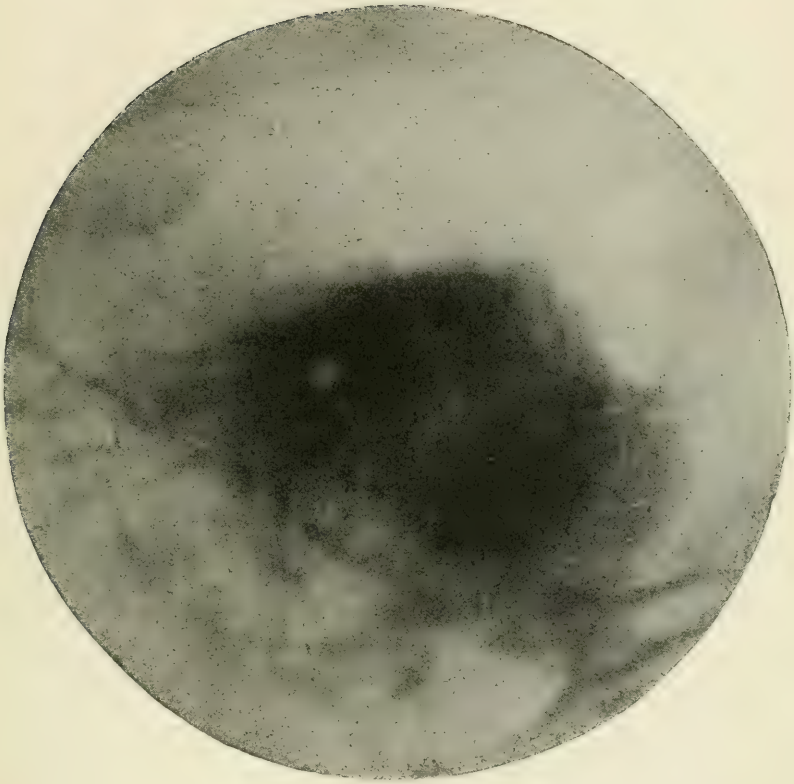


No. 12 (Right).

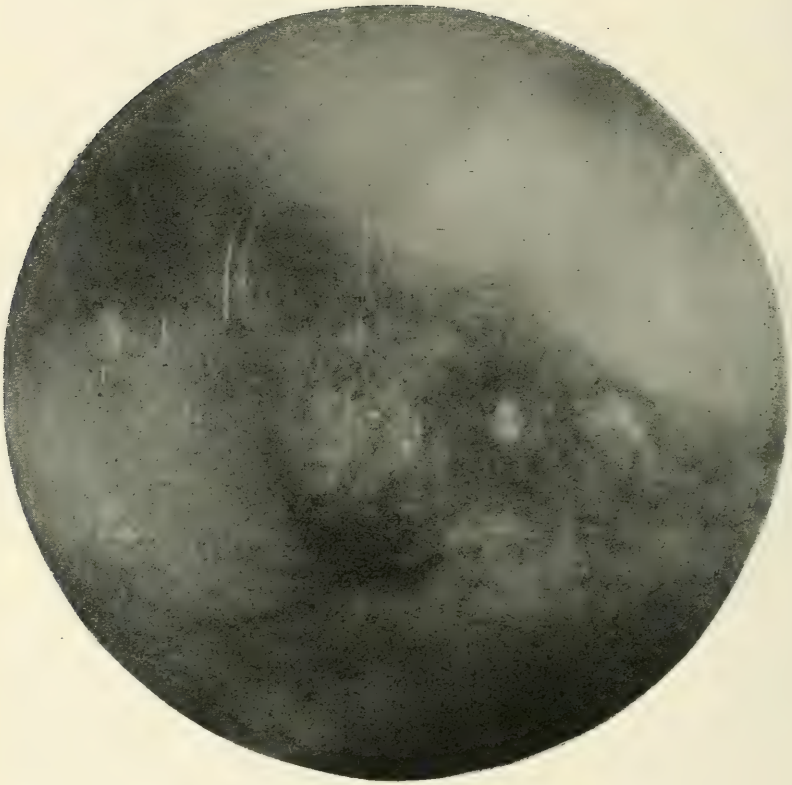


No. 13 (Left).

No. 13 (Right).



No. 14 (Left).



No. 14 (Right).

Discussion.

Dr. E. B. Dench, New York City, thought that while X-ray pictures might be of value in the diagnosis of mastoid conditions, it required an expert to read them. A man might be an expert in reading the pictures of one radiographer and not of another. He was connected with several hospitals, and could read the X-ray pictures of one radiographer without difficulty, because he was familiar with the position of the head in which the pictures were taken. The pictures of another radiographer looked a great deal better, but he could not read them as well as he could those of the other. A radiographer who excelled in abdominal work might not be able to do mastoid work at all. He cited an instance in which a mistake occurred. A radiograph was taken in a case in which the right mastoid was involved. The radiographer made the diagnosis of involvement of the left. He opened the right, and verified the clinical diagnosis. The value of the radiograph had been demonstrated on his service without question. In a case of acute otitis media in which he did not know what the progress of the case would be, he had an X-ray taken, waited four or five days, had another taken, waited again, and had the third taken. By reason of these pictures he could forecast pretty thoroughly what would be found. It was impossible, in the early stages, to tell whether the condition was beginning sclerosis or something else. He recalled one case in which there was almost total absence of symptoms and which he thought was going on to sclerosis. He followed the case for six or seven months, and the diagnosis which the radiologist made of beginning sclerosis was confirmed. In a number of cases the otitis media had occurred several months before the patient came under his observation, and had recurred. In one case in which there was trouble with the hearing, the X-ray showed what was supposed to be an extradural abscess. Upon operation the abscess was found exactly where the X-ray had indicated.

Dr. George E. Shambaugh, Chicago, had found the radiograph of assistance in doubtful cases. He cited a case of a thick-necked man, where a swelling developed below the mastoid, in the presence of an acute otitis media. The case had been diagnosed by another physician as an infected cervical gland. There was no tenderness over the mastoid but the radiograph disclosed a broken-down mastoid process. The condition was one of a Bezold mastoiditis. In just such cases he finds the X-Ray of great value. He does not see the necessity for having a radiogram made a routine to be applied to all cases, in as much as the clinical examination in most cases gives all the data required for a positive diagnosis. He finds the X-Ray examination of greater value than the transillumination of the mastoid.

Dr. Frank T. Hopkins, New York City, emphasized the importance of X-ray work as a routine procedure, because the more one employed it the more familiar one became with just the points it was desirable to know. The work at the New York Eye and Ear Infirmary had established the value of routine X-ray work. Another point of special value in X-ray work was that it showed the relative position of the sinus to the posterior wall.

Dr. Harris P. Mosher, Boston, agreed with Dr. Shambaugh as to the routine use of the X-ray. It was impossible to tell by transillumination or by X-ray whether a given appearance was the result of ordinary otitis media or a breaking down of the mastoid. It might be, as Dr. Hopkins had said, that the finer points would become more familiar as the result of routine X-ray work, and for that reason he had continued to employ it.

Dr. John B. Rae, New York City, was unable to report uniform success in the use of the X-ray in doubtful cases. Where the clinical evidence is unmistakable, little is to be gained by the use of the X-ray, and it has been his experience, to find in a large proportion of the cases which

are doubtful clinically, the X-ray picture has been of little assistance. In many of our cases, the observer is himself satisfied of the necessity, sometimes urgent necessity, of operation, while the subjective symptoms are so little in evidence that the patient and his friends are hard to convince of this real necessity. The X-ray picture may show such a marked difference between the two mastoids as to turn the scale as far as the lay-observers are concerned in favor of operation.

Dr. Ernest de Wolf Wales, Indianapolis, Ind., said, with reference to the relationship of the sinus to the external auditory canal, that the sinus might be projected and apparently very near the auditory canal, whereas in reality it might be very far from it. If one did not know how the picture was projected one could tell nothing about it. The auricle too might obscure the picture.

Dr. Amberg, in closing the discussion, said that if an aurist of such ability and experience as Dr. Dench, was so materially helped in his diagnosis by the X-ray examination, it was justifiable to recommend this kind of examination as a routine measure.

SERIAL

RF
1
A55
v.13

American Otological Society
Transactions

GERSTS